Candidature au diplôme d'Habilitation à Diriger des Recherches

déposée par

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http://ur-agirs.Cirad.fr / www.rp-pcp.org

À l'Ecole Doctorale G.A.I.A.

(BIODIVERSITE, ALIMENTATION, AGRICULTURE, ENVIRONNEMENT, TERRE ET EAU)

DE L'UNIVERSITE MONTPELLIER II

Sur le thème "Ecologie de la transmission infectieuse
dans les systèmes multi-hôtes
des socio-écosystèmes africains "

Remerciements

Les deux piliers indispensables à la bonne santé professionnelle du chercheur sont sa famille et ses amis, et son entourage professionnel “du premier cercle”. J’ai la chance de pouvoir compter sur les deux sans même une distinction claire puisque je considère beaucoup de mes collègues comme des amis voire certains comme membres de la famille.

Ma vie de famille a largement été influencée par ma carrière professionnelle puisque mes deux filles sont toutes deux nées au Zimbabwe et l’équation de leurs vies se construira forcément en fonction de cette expérience. Carole m’a suivi au prix de sa propre carrière vétérinaire mais son dynamisme et son hyper activité sociale lui ont toujours permis de vivre pleinement sa vie active ; et le défi de notre installation au Mozambique ne sera qu’un nouveau tremplin vers une reconversion voulue et excitante. Mon asociabilité est tous les jours débordée par la vie que tu nous organises et j’en suis ravi. Tu m’impressionneras toujours ! Ma sœur et toute la famille Laparre est venue nous rejoindre au Zimbabwe en 2008 : merci ! Ces belles années au Zimbabwe auront marquées nos deux familles. Quant à mes parents, récents émigrés sur les côtes sud de l’Espagne mais que je ne plains pas, vous êtes toujours là pour nous et si dire que sans vous je ne serais pas là est une évidence, il faut souligner l’inné mais aussi l’acquis qui m’ont mis sur les rails de ma carrière.

Nouveau paragraphe, mais on reste dans la famille: Michel (et Sophie), témoin de tellement d’évolutions positives dans ma vie personnelle et professionnelle… Pour l’aspect professionnel, non seulement témoin mais grand acteur ! Il nous reste encore un long parcours scientifique à construire que nos nouvelles expériences à venir alimenteront je l’espère. Toujours dans la famille, Eve et Vlad, vous arrivez quand ? Ici ou ailleurs mais pas trop loin quand même… Et puis tous les amis éconapiens et agirois de la Zimbabwe connexion ou non, que j’ai appris à connaître au fil de nos projets ou affectations. C’est un plaisir de travailler avec vous et de partager plus que du travail de temps en temps. Il me reste encore beaucoup de chercheurs à mieux découvrir à AGIRs et au Cirad plus largement et avec qui je sens que ce sera plus que du simple travail. Cet environnement professionnel ciradien est un bijou qu’il ne faudra pas laisser filer. Finalement, trois mentions spéciales : à ceux de Hwange avec qui on partage aussi plus que de la collaboration formelle Cirad-CNRS ; aux assistantes d’AGIRs (une petite dédicace à Catherine) qui assurent nos arrières et participent à la vie scientifique et collective au delà de leur mandat; et aux nouvelles têtes comme Arthur et Hugo qui doivent rester dans le paysage !

C’est une chance incroyable que j’ai d’évoluer dans un contexte personnel et professionnel aussi dense et dédié. Je le sais et je m’en rends compte. Merci à tous. Et aussi à vous, rapporteur et membres du jury, connus ou inconnus pour ce temps que vous n’avez pas mais que vous consacrez quand même à mes introspections scientifiques.

I. Content

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IV. Curriculum vitae

A. Civil status and contact details

Alexandre CARON

Citizenship: French
Civil Status: Married, 2 daughters
Birth Date: 5 of August 1975 in Paris (XIII^{e})

Professional contact details:
1/ Cirad/RP-PCP, Faculdade de Veterinária - Universidade Eduardo Mondlane, Avenida de Moçambique Km. 1,5 - Caixa Postal 257, Maputo 01009, Moçambique; Cell: + (258) 84 464 06 39

2/ Cirad, UPR AGIRs, "Animal and Integrated Risks Management), TA 30/F, Campus international de Baillarguet, 34398 Montpellier Cedex 5, France, Cell: + (33) 4 67 59 58 00

Email: alexandre.caron@cirad.fr

B. Academic and professional pathway

2010 - on-going: Cirad researcher, UPR AGIRs, "Animal and Integrated Risks Management), posted in **Zimbabwe** from 2010 to 2014 and **in Mozambique since 2015**.

2006 - 2011: PhD (submitted in 2010 and defended in 2011), Mammal Research Institute, University of Pretoria, Pretoria, South Africa: "Describing and understanding host-pathogen interaction at the wildlife/domestic interface ". Supervisors: Prof. Elissa Cameron (MRI), Prof. David Cumming (University fo Cape Town), Dr. Michel de Garine-Wichatitsky (Cirad), Dr. Serge Morand (CNRS). Based in **Zimbabwe**.

2005 - 2007: Cirad expert for FAO TCP Avian Influenza project. Set-up and implementation of Wild birds' surveillance missions & training for Avian Influenza in **Mali, Chad, Niger, Malawi, Ukraine, Zambia, South Africa**)

2004: Pan African Control of Epizootics (PACE) wildlife expert for Cirad, based in **Kenya**. Technical assistant based at AU-IBAR office (Nairobi); Support in wildlife epidemiology and capture in **Kenya, Chad, Ethiopia, Tanzania, Cameroon, Ghana**.

2002 - 2003: Consultant epidemiologist for Société Générale de Surveillance (SGS) for Rift Valley Fever control in the Horn of Africa (4 months, **Ethiopia, Somliland, Dubai, Djibouti**).

2002: First contract with Cirad to work on the proposal of the EDEN project (Emerging Disease European Network), **France**.

2001: MSc, "African Mammalogy: Conservation and Management of Diversity", Mammal Research Institute, University of Pretoria, Pretoria, **South Africa**.

2001: DESS "Animal Production in Tropical Areas", Cirad, Montpellier, **France**.

1996-2001: Diploma in Veterinary Medicine (DVM), Veterinary School of Maisons-Alfort, Maisons-Alfort, **France**.

C. Scientific prizes

"AHEAD Beyong Boundaries" Transboundary & Emerging Disease Journal Awards, *July 2009*. For researchers and managers conducting promising work at the interface between wildlife health, domestic animal health, and human health and livelihoods.

First Price "Student Award", *May 2002*, Heidelberg, Germany. European Wildlife Disease Association Meeting; Presentation: "Impact of Bovine Tuberculosis on African Buffalo in Kruger National Park".

D. Summary of publication and reviewing activity

1. Summary of publication activity

The details of publications (journals, impact factors, co-authorship) are provided in the section "List of publications and communications".

In November 2015, Google Citation estimated my **h-index to be 15 and my i10 to be 21**. These indices are known to be higher than for other platforms but take into account part of the literature also important for Cirad.

In summary, my publication list is composed of:

Table 1: Publication summary

Publication type	Total	1st Author	2nd Author	Last Author	Including student
Journal & ISI IF*	33	9	8	1	5
Journal no IF	10	3	-	3	1
Book chapter	5	1	-	2	0
Total	**48**	**13**	**8**	**6**	**6**

**ISI Impact Factor*

Eight articles are currently submitted (including 2 with students as co-authors).

I have participated as well to 62 oral communications and around 16 poster presentations in international and regional conferences. Numerous oral presentations during technical committees, trainings and local meetings are not presented here.

To have an updated list and access to my publications, one can consult webpages where I regularly add material about my publications:

Cirad webpage
Research gate
Google citations

2. Summary of reviewing activity

Publication reviewing and journal editing is an integral part of researchers' activity. However, until recently, it was difficult to provide indices or list of works. Recently, a new website provides a way to be visible on this kind of activity and I have registered and built my personal reviewer page here:

Publon

My reviewer activity can be summarised as follow:

Table 2: Number of reviews achieved per journal

Journal	Nb of review*
Proceedings of the Royal Society B	2
Journal of Applied Ecology	1
Ecological Applications	2
Landscape Ecology	1
Veterinary Research	3
PLoS One	4
Preventive Veterinary Medicine	2
Ecological Modelling	1
Ecology & Society	1
EcoHealth	1
Avian Diseases	1
Infection, Ecology & Epidemiology	1
Journal of Wildlife Management	1
Tropical Animal Health and Production	2
Journal of Epidemiology and Global Health	2
Pathogens	1
Polish Journal of Environmental Studies	1
TOTAL	**27**

** Including second review of revised manuscript*

The totality of these reviews have been completed since the end of 2009, including 6, 6 & 7 for 2013, 2014 and 2015, representing the **majority of the reviews in the last 3 years**.

Since 2013, I am also an **editor for the Ostrich Journal**. This position represents on average 1 article to edit every year (I am receiving all articles dealing with pathogens/diseases in wild birds).

E. Involvement in project proposals & implementation

In this list, I have added at the end of each paragraph a note (i.e. "proposal", "implementation", "coordination" and "P.I.", "co-P.I.") indicating respectively if I participated in the proposal writing, project implementation and/or coordination, "P.I." and "co-P.I." indicating if I was the main researcher involved in this activity (or the co-main investigator). "M" and "K" in front of a monetary unit (e.g. "$" or "€") indicate respectively millions and thousands.

1. Most recent projects, in Mozambique

SAIRLA Small-Ruminant Diseases, 750K£ (DFID), proposal
SAIRLA ICP Burkina/Tanzania, 750K£ (DFID), proposal
African Swine fever Ecology in southern Africa, >1M€ (NSF), proposal
Stork and antibiotic resistance spread, 100K$ (Gates Foundation), proposal

ANR Anihwa IUEPPR - 2014-on-going, 1.3M€ (ANR), coordinated by Pirbright Institute, "Improved understanding of the epidemiology of peste-des-petits ruminants", *implementation*

Platforme ESA - 2014-on-going, (Cnesa), ESA, coordinated by ANSES (Veille Sanitaire Internationale) - "French Platform of epidemiosurveillance in animal health", *implementation*

2. Since 2006, based in Zimbabwe

RP-PCP, 2007-on-going, around 850K€ (MAE), RP-PCP, coordinated by Cirad, "Research Platform - Production & Conservation in Partnership" - Co-coordinating the platform since 2012, Secretary Coordinator and Animation of Cirad activities within the platform since 2015, *coordination & implementation*

EU-DREAM - 2014-on-going, 2M€ (EU), DREAM, coorindated by Cirad - "Delivering Innovation and technology through the REinforcement of Agricultural and Multidisciplinary research capacity for the benefits of small-scale farmers in TFCAs" - *proposal*

EU-GeosAf - 2014-on-going, 1M€ (EU), GeosAf, coordinated by Cirad - "Geomatic technology transferred to animla health services in southern Africa", *co-P.I. proposal*

FSP RenCaRe - 2012-on-going, 500K€ (MAE), RenCaRe, coordinated by Cirad "Strengthening of research capacity for the management of protected area and their periphery in southern Africa", support to RP-PCP - *implementation* (student supervision)

PEPS, 2012-2013, 10K€ (Uni. of Montpellier), coordinated by CNRS - "Comprendre les maladies émergentes et les épidémies: modélisation, évolution, histoire et société" - *proposal & implementation*

ANR SAVARID - 2011-on-going, 850K€ (ANR), SAVARID, coordinated by CNRS - "Increased Aridity in Hwange Socio-Ecological System" - *proposal, implementation & coordination* (P.I. one workpackage "Animal health")

BUCATIN - 2010-on-going, 55K€ (FAO, Cirad), BUCATIN, coordinated by Cirad - "Buffalo/cattle interaction and shared disease burden in the GLTFCA" - *P.I. proposal, implementation & coordination*

FSP-GRIPAVI - 2007-2011, 3.6M€ (MAE), GRIPAVI, coordinated by Cirad - "Ecology and epidemiology of avian influenza in southern countries". This project was implemented in 6 countries and funded my PhD - *proposal, implementation & coordination (P.I. for Zimbabwean site)*

CORUS FMD - 2008-2010, 70K€ (CORUS), CORUS, coordinated by Cirad - "Development of an epidemiological network for monitoring the dynamics of FMD within the GLTFCA" - *implementation*

EU-PARSEL - 2008-2011, 1.9M€ (EU), PARSEL, coordinated by Cirad - "Public-Private-Community Partnerships to improve food security and livelihoods in

the South East Lowveld and Mid Zambezi Valley", - *proposal & implementation* (workpackage "Animal Health")

3. Prior to 2006

TCP FAO Avian Influenza - 2005-2007, 3M$ (FAO), coordinated by Cirad - FAO TCP project aiming at investigating the role of wild birds in the epidemiology of Avian Influenza in Europe and Africa - ***implementation***

PACE - 2004, >40M€ (EU), coordinated by AU-IBAR - Wildlife Expert for the Pan-African Control of Epizootics EU project (Cirad contract), based in Nairobi. This project aims at an African eradication of rinderpest, a goal achieved in 2011 - ***implementation***.

EDEN - 2002-on-going, (EU), EDEN, coordinated by Cirad - short-term contract with Cirad - ***proposal***.

Of course, the list of successful project proposals hides a much longer list of projects not funded that have been nonetheless submitted but that cannot be reported here.

F. Student supervision, degree reviewing and training

1. Postgraduate students

PhD

MUNDAVA, Josphine (2010 - on-going). "The Structure and Dynamics of a Waterbird Assemblage on two Large Dams". PhD, National University of Science and Technology, Bulawayo, Zimbabwe. Main supervisor : Prof. P. Mundy (NUST); co-supervisors : Drs A. Caron (CIRAD), M. de Garine-Wichatitsky (CIRAD) & N. Gaidet (CIRAD).

Without minimizing the role of Josphine's other supervisors, I can say that I represented Josphine's principal supervisor. She realised her MPhil (2008-2010) then her PhD in the framework of the GRIPAVI project in the Zimbabwean site that I was coordinating. Her PhD fit with mine and she built her protocols in relation to mines and I trained her during our fieldwork to different techniques (bird sampling / catching / identification). I have been intensely involved in the design of her thesis and her first papers. Josphine is now a lecturer at NUST and she has been struggling to conclude her PhD. We hope that by the end of the 2015, she will have defended. ***We collaborated on 5 articles*** *with IF and 1 without IF together, including 1 with her as 1st author and 2 with me as 1st author. Her second 1st author article is currently under review after major revision in Ibis Journal.*

MUKARATI, Norman (2014 - on-going). "The epidemiology and ecology of Bacillus anthracis infections (anthrax) in wildlife/livestock interface areas in Zimbabwe". PhD, University of Zimbabwe, Harare, Zimbabwe. Main supervisor: Prof. D. Pfukenyi & Prof. G. Matope (Uni. of Zimbabwe); co-

supervisors: Drs A. Caron (Cirad), M. de Garine-Wichatitsky (Cirad), Dr. H. van Heerden (Uni. of Pretoria).

Norman's PhD started at the beginning of 2014. I act strictly as a co-supervisor, helping in the design of his proposal and its protocols. We are in regular contact and I try to meet him whenever I go back to Zimbabwe (3 to 4 times a year). We have not yet published together.

MSc/MPhil

BORT-CABALLE, Anna (2015), "Contraints on animal production in the Mozambican part of the GLTFCA: animal health, risks and management". RenCaRe Project. MSc (M2), University of Montpellier II. Main supervisors: Drs. A. Caron (Cirad) & J. Fafetine (UEM).

MARTINS, Irisalda (2015), "Animal production and resource access (water/grazing) in the Mozambican part of the GLTFCA". RenCaRe Project. MSc, Universidade Edouardo Mondlane, Faculdade de Veterinaria. Main supervisors: Dr. C. Garrine (UEM) & A. Caron (Cirad); co-supervisor: Dr. R. Ducrot (Cirad).

SAMAPODISA, Omphile (2014 - on-going), "Land use change and cattle posts influence on wildlife distribution across different land uses in the Chobe enclave". RenCaRe Project. MPhil, Okavango Research Institute. Main supervisors: Drs R. Fynn (ORI), G. Masunga (ORI), L. Rutina (ORI); co-supervisors: Dr A. Caron (Cirad), Dr M. de Garine-Wichatitsky (Cirad).

MASSOT, Méril (2014), "Diversity of Escherichia coli populations and antibioresistance at buffalo/cattle interfaces in southern Africa: individual and spatio-temporal variability". MSc (M2), University of Montpellier II. Main supervisors: Drs. A. Caron (Cirad) & E. Denamur (INSERM 1137).

Méril is currently finishing a second MSc in Biostatistics and she should start her PhD with Dr. E. Denamur as her main supervisor in 2015 or 2016. She should be able to publish 1 article as 1st author and is a co-author on another article resubmitted after revision (see just below).

MERCAT, Mathilde (2013), "Escherichia coli populations sharing and antibioresistance gradient at a buffalo/cattle interface in southern Africa". MSc (M1), University of Montpellier II. Main supervisors: Drs. A. Caron (Cirad) & E. Denamur (INSERM 1137). *One article as 1st author (and me as last author) resubmitted after major revision.*

MANGEYA, Danai (2013 - on-going), "Intestinal macroparasite diversity and burden in cattle population with contrasted surface water availability and wildlife interactions". ANR SAVARID. MPhil, University of Zimbabwe, Faculty of Veterinary Medicine. Main supervisor: Profs D. Pfukenyi (UZ), G. Matope (UZ); co-supervisors Drs A. Caron (Cirad), M. de Garine-Wichatitsky (Cirad), Prof. G. Vassilev (UZ).

Danai used to have a lecturer position at the Faculty of Veterinary Medicine of UZ. His position was nit renewed due to financial constraints.

Since one year, Danai is struggling to properly write his thesis as he is currently working full time in a veterinary clinic. We hope that by the end of 2015 or mid-2016, he will be able to defend his thesis and publish at least one paper as 1st author.

ZISHIRI, Sarudzai (2012), "Understanding Socio-Economic Factors that Influence Local People's Capacity to Access and Manage Cattle in Sengwe Communal Land". MSc, University of Zimbabwe, Centre for Applied Social Sciences. Main supervisor : Dr B. Mukamuri (UZ); co-supervisors : Dr M. de Garine-Wichatitsky (Cirad) & Dr A. Caron (Cirad).

CHIGWENESE, Leoba (2010 - on-going), "Permeability of selected fences to wildlife and livestock in the South East Lowveld of Zimbabwe". MPhil, UZ/Department of Geography and Environmental Sciences. Main supervisor : Dr A. Murwira (UZ); co-supervisors : Dr M. de Garine-Wichatitsky (Cirad) & Dr A. Caron (Cirad). One paper submitted.

Leoba is waiting for her article to be accepted in order to defend her thesis (at Uni. of Zimbabwe, to defend a MSc or a Mphil at least one article must be accepted).

2. Other degrees

HUNGWE, Last (2014), "A retrospective study on the transmissible diseases among wildlife, livestock and humans in the wildlife/livestock/human interface of Hwange and its surrounding districts of Zimbabwe for the period 1 January 2008 to 31 December 2013". BVSc, University of Zimbabwe, Faculty of Veterinary Science. Main supervisors: Prof. D. M. Pfukenyi (UZ), Drs N. Mukarati (UZ) & A. Caron (Cirad). *One article in preparation*.

BULEYA, Blessing (2012), "Distribution and biometric measurements of African Jacana in Chivero and Manyame Dams between 2007 and 2010". BSc, NUST. Main supervisors: Prof. P. Mundy (NUST) & Dr A. Caron (Cirad).

3. Implication on other MSc/MPhil/PhD

Since 2008, I have also been involved with several students (for some of them when I was still a PhD student) without being formally in their supervision team. However, I feel that our scientific collaboration requires that I mention them, just to give an indication of our scientific production.

MIGUEL, Eve (2009-2012). "Pathogens transmission from wild to domestic herbivores in Zimbabwe". PhD, Université Montpellier II, Doctoral School SIBAGHE. Main supervisor: Dr M. de Garine-Wichatitsky (Cirad); co-supervisors: Dr H. Fritz (CNRS) et Dr T. Boulinier (CNRS). ***Five articles*** *with IF including 3 with Eve as 1st author and two with me as 1st author; 3 articles submitted and 1 in prep.* Eve is currently doing a post-doc with MIVEGEC.

ZENGEYA, Fadzai (2009-2014). "Understanding the distribution of cattle at the livestock-wildlife interface using real-time Global Positioning Systems (GPS) and satellite remotely sensed data". PhD, UZ/Department of Geography and

Environmental Sciences. Main supervisor: Prof A. Murwira (UZ); co-supervisor: Dr M. De Garine-Wichatitsky (Cirad). ***One book chapter*** *and one article recently accepted*. Fadzai is now lecturer at UZ.

ZVIDZAI, Mark (2008- 2012). "Waterhole use by wild and domestic ungulates: exploring temporal niche use and associated competition and pathogen transmission potentials". MPhil, UZ/Department of Geography and Environmental Sciences Main supervisor: Dr A. Murwira (UZ); co-supervisor : Dr M. de Garine-Wichatitsky (Cirad). ***One article*** *with Mark as 1st author*.

DUBE, Timothe (2009-2010). Permeability of selected fences to wildlife and livestock in the South East Lowveld of Zimbabwe. MPhil, UZ/Department of Geography and Environmental Sciences. Main supervisor: Dr A. Murwira (UZ); co-supervisor : Dr M. de Garine-Wichatitsky (Cirad). ***One book chapter*** *with Timothe as 1st author*. Timothe is doing is PhD in the USA.

GOMO, Calvin (2007-2010). "A survey of tuberculosis and brucellosis in wild and domestic animals in the SEL of Zimbabwe". MPhil, UZ/Faculty of Veterinary Sciences. Main supervisor: Dr D. Pfukenyi (UZ); co-supervisor : Dr M. de Garine-Wichatitsky (Cirad). ***Four articles*** *with IF including 2 with Calvin as 1st author and one as me as 1st author*. Calvin is now lecturer at Chinoyi University.

4. Reviewer for degrees and carrier upgrades

2014 - Peer-review of the carrier of Dr. Bernard Bett (ILRI). ESPA Early Career Research Fellowship 2013 (Ecosystem Services for Poverty Alleviation).

2014 - Peer-review of the carrier of Mr. Mduduzi Ndlovu for a carrier upgrade. University of Witwatersrand.

2010 - Peer-review of Christine Moore MSc thesis "Understanding Highly Pathogenic Avian Influenza Outbreaks in the Western Cape Ostrich Industry : Did Network Dynamics Enhance Vulnerability?". University of Cape Town.

5. Involvement in training activities

2014: Cirad, Montpellier, France. "Master class One Health " organised within the Sea-EU-NET project. Half a day of teaching and half a day of of discussion on "Disease Ecology at wildlife/livestock interfaces".

2013: Universidad de Castilla-La-Mancha/IREC, Cuidad Real, Espagne. "One Heath Course" organised within the ANTIGONE (EU-FP7) project. Three hours of teaching (+ 1 hour of exercises) in collaboration with M. de garine-Wichatitsky on "Epidemiological interactions at wildlife-livestock interfaces".

2013: University of Pretoria, South Africa. "1st One Health Summer School" organised by the Faculty of Veterinary Science. One presentation on on-going work and facilitation of the course during a one-week trip.

G. Collective responsibilities

2015 - on-going: Elected "Secretary Coordinator of the Research Platform "Production & Conservation in Partnership" (RP-PCP) by the Steering Committee.
2015 - on-going: Nominated as "Animateur du Dispositif Prioritaire RP-PCP" for Cirad.
2012 - on-going: Elected webmaster of the RP-PCP by the Steering Committee.
2012 - 2015: Nominated as "co-Animateur du Dispositif Prioritaire" RP-PCP for Cirad.
2010 - 2015: Elected as "Co-coordinator" of the Research Platform "Production & Conservation in Partnership" (RP-PCP) by the Steering Committee.

H. Expertise and evaluation

1. Expertise

2013 - FAO SPINAP Wildlife training, 1 week, Zimbabwe. Training of SADC representative on wildlife sampling and eco-epidemiology.
2010 - OIE Wildlife focal points training, 1 week, Mali. Wild bird capture, eco-epidemiology, wildlife/livestock interface.
2008 - FAO Great Lakes Project, 3 weeks, Rwanda, Burundi, RDC. Training expertise on wild bird and avian influenza ecology. Wild bird capture, handling, sampling.
2007 - ANR Mobility, 3 weeks, Niger. Buffalo capture and sampling.
2005-2007 - FAO Wildlife Expert/Avian Influenza Emergency Plan – technical & training expertise, 6 months, Mali, Chad, Niger, Malawi, Ukraine, Zambia, South Africa. Set-up an dimplementaiton of wild bird's surveillance for Avian Influenza; Expert training in malawi, Ukraine & South Africa.
2005 - GTZ-SATEC consultant for AU-IBAR, 1 month, Ghana. Training of regional and national team in wildlife capture & epidemiology.
2005 - EWDA (European Wildlife Disease Association), 1 month, France. Electronic conference " Wildlife Infectious Emerging and Re-emerging Diseases in Europe"; 80 participants; organisation and moderation.
2005 - LEAD-FAO electronic conference moderation, 1 month, France. Electronic conference "Interface wildlife/livestock: the sanitary risk"; 450 participants, moderation.
2004 - PACE Regional Wildlife Expert, 11 months, Kenya (based), Chad, Ethiopia, Tanzania, Cameroon, Ghana. Technical assistant based at AU-IBAR. WOrk on Rinderpest, ASF & FMD.
2002-2003 - Consultant epidemiologist for SGS (Société Générale de Surveillance), 4 months, Djibouti, Dubai, Ethiopia, Somaliland, Switzerland. Technical assistant on Rift Valley Fever Trade.

2. Project evaluation

2014 - Peer-review for the project proposal "Social, Economic and Environmental Drivers of Zoonoses in Tanzania (SEEDZ)". Standard proposal for BBSRC call, UK.

2011 - Peer-review for the project proposal "HDSS in livestock". National Research Foundation South Africa (NRF).

I. Conference & workshop organisation

2014 - RP-PCP/AHEAD joint conference ", Hwange, Zimbabwe. Member of the scientific and organisation committee.

2014 - African Buffalo Symposium, Paris, France. Member of the scientific and organisation committee.

J. Member of boards and committees

2014 - on-going: Member of the advisory board of the AHEAD GLTFCA Initiative.
2014 - on-going: Co-coordinator of the African buffalo Interest Group (AfBiG), a group of the IUCN Antelope Specialist Group.
2007 - on-going: Member of the IUCN Veterinary Specialist Group
2010 - on-going: Member of the IUCN Pigs, Peccaries & Hippos Specialist Group
2004 - 2012: Member of the Africa and Middle East Section of the Wildlife Disease Association
2002-2004: Member of the European Section of the Wildlife Disease Association

V. Detailed list of publications

A. A few statistics on my publications

In November 2015, my list of publications includes 43 peer-reviewed publications, including 31 different journals and 5 book chapters.

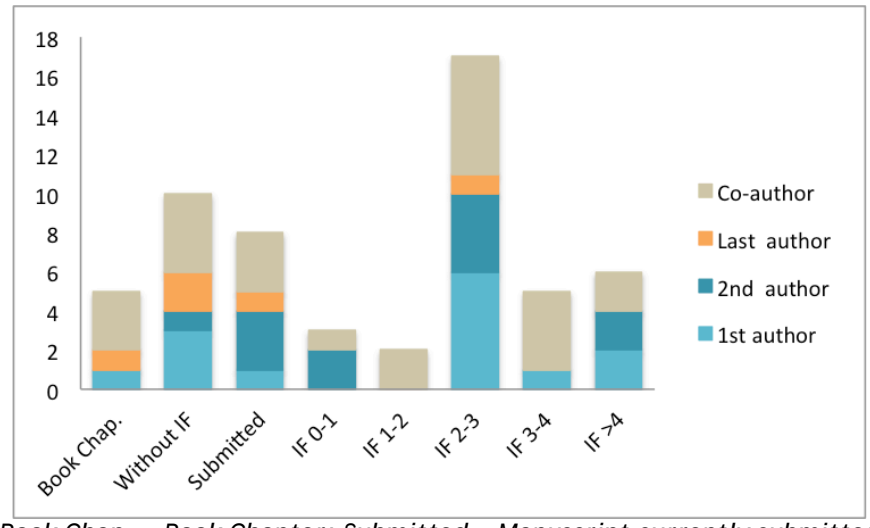
Table 3: Journals in which I published

Journal name	Nb of articles	IF2014
Acta Tropica	1	2,27
Annals of the New York Academy of Sciences	1	4,38
BMC Evolutionary Biology	1	3,37
Bulletin de l'ANSES	1	na
Comparative Immunology and Microbiology of Infectious Diseases	1	2,01
EcoHealth	3	2,45
Ecological Applications	1	4,09
Ecology & Society	1	2,77
Ecosphere	2	2,25
Emerging Infectious Diseases	3	6,75
EMPRES - Animal Health	2	na
Epidemiology & Infection	4	2,53
Game & Wildlife Science	1	na
Honeyguide	2	na
Infection, Genetics & Evolution	1	3,01
International Journal of Development & Sustainability	1	na
Journal of Wildlife Diseases	1	1,35
Landscape Ecology	1	3,5

Onderstepoort Journal of Veterinary Research	1	1,26
Ostrich	1	0,41
Parasitology	1	2,56
PLoS ONE	2	3,23
Preventive Veterinary Medicine	1	2,17
Proceedings of the Royal Society B	1	5,05
Remote Sensing Applications: Environment and Society	1	na
Revue d'Anthropology des Connaissances	1	na
Revue Scientifique et technique de l'OIE	1	0,91
Sécheresse	1	na
Transboundary & Emerging Diseases	1	2,94
Tropical Animal Health & Production	1	0,82
Veterinary Research	2	2,81
Total	43	

In Figure 1, I have displayed the Journal Impact factor in relation to my position in the authorship. The figure includes as well the book chapters, the journals without IF and the manuscripts currently submitted.

Figure 1: Authorship & Impact Factor of publications



Book Chap. = Book Chapter; Submitted = Manuscript currently submitted

Of these publications, only 6 have been co-authored with students that I have supervised. However, this number can be completed by 2 others:

- out of the 8 manuscript currently submitted, 2 are led by 2 of the students I supervised (and me as 2nd and last author).
- if I include the students that I have listed in IV.F.3 (students from the RP-PCP that I was not formally supervising but with who I have interacted a lot during their thesis), the number rises to 20, plus 5 from the 8 submitted manuscripts.

This remark highlights the fact that, as I have done my PhD quite late, I was not able to get involved with student supervision very early in my carrier. However, the data presented here serves to demonstrate that I collaborate very efficiently with other researchers, including students.

B. Detailed list of publications

1. Peer-reviewed articles in Journals with an ISI IF

Names underlined like this represent direct supervision, and like this represent undirect supervision as defined previously (IV.F.3).

A33. HELLARD E, CUMMING GS, **CARON A**, COE E, PETERS J

Testing epidemiological functional groups to predicts avian haemosporidia patterns in southern Africa

Ecosphere, **2015**, in press.

A32. **CARON, A.,** CORNELIS, D., FOGGIN, C., HOFMEYR, M., DE GARINE-WICHATITSKY, M

Transboundary Conservation Areas, African Buffalo movements and Animal Diseases

Emerging Infectious Diseases, **2015**, in press.

A31. **CARON A**, CAPPELLE J, CUMMING G, DE GARINE-WICHATITSKY M, GAIDET N

Bridge hosts, a missing link for disease ecology in multi-host systems

Veterinary Research, **2015**, 46: 83

A30. CUMMING GC, ABOLNIK C, **CARON A**, GAIDET N, GREWAR J, HELLARD E, HENRY D, REYNOLDS C

A review of regional variation in avian influenza dynamics: insights from a social-ecological systems perspective

Landscape Ecology, **2015**, 30(6): 963-985

A29. CAPPELLE J, **CARON A**, SERVAN DE ALMEIDA R, GIL P, PEDRONO M, MUNDAVA J, FOFANA B, BALANÇA G, DAKOUO M, OULD EL MAMY AB, ALBONIK C, MAMINIAINA O, CUMMING GS, DE VISSCHER MN, ALBINA E, CHEVALIER V, GAIDET N

Empirical analysis suggests continuous and homogeneous circulation of Newcastle Disease virus in a wide range of Wild Birds species in Africa

Epidemiology and Infection, **2015**, 143(6): 1292-1303

A28. SMITZ N, CORNELIS D, CHARDONNET P, **CARON A**, DE GARINE-WICHATITKSY M, JORI F, MOUTON A, LATINNE A, PIGNEUR L-M, MELLETTI M, KANAPECKAS KL, MARESCAUX J, LOPES PEREIRA C, MICHAUX J

Genetic structure of fragmented southern populations of African Cape buffalo (Syncerus caffer caffer)

BMC Evolutionary Biology, **2014**, 14: 203.

A27. MIGUEL E, BOULINIER T, DE GARINE-WICHATITSKY M, CARON A, FRITZ H, GROSBOIS V

Characterising African tick communities at a wild-domestic interface using repeated sampling protocols and model

Acta Tropica, **2014**, 138: 5-14

A26. JORI F, **CARON A**, THOMPSON PN, DWARKA R, FOGGIN C, DE GARINE-WICHATITSKY M, HOFMEYR M, VAN HEERDEN J, HEATH L

Characteristics of Foot-and-Mouth Disease Viral Strains Circulating at the Wildlife/livestock Interface of the Great Limpopo Transfrontier Conservation Area

Transboundary & Emerging Diseases, **2014**, DOI: 10.1111/tbed.12231

A25. **CARON A**, GROSBOIS V, ETTER E, DE GARINE-WICHATITSKY M

Bridge hosts for Avian Influenza viruses at the wildlife/domestic interface: an eco-epidemiological framework implemented in southern Africa

Preventive Veterinary Medicine, **2014**, 117: 590-600

A24. SMITZ N, BERTHOULY C, CORNELIS D, HELLER R, VAN HOOFT WF, CHARDONNET P,

CARON A, PRINS HHT, VAN VUUREN BJ, DE IONGH HH, MICHAUX JR

Pan-African Genetic Structure in the African Buffalo (Syncerus caffer): Investigating Subspecies Divergence

PLoS One, **2013**, 8(2): e56235.

A23. MIGUEL E, GROSBOIS V, **CARON A**, CORNELIS D, BOULINIER T, FRITZ H, FOGGIN C, MAKAYA P, TSHABALALA P, DE GARINE-WICHATITSKY M

Contacts and foot and mouth disease transmission from wild to domestic bovines in Africa

Ecosphere, **2013**, 4(3): 51

A22. MIGUEL E, GROSBOIS V, BERTHOULY-SALAZAR C, CARON A, CAPPELLE J, ROGER F

Meta-analysis of Observational Epidemiological Studies of Newcastle Disease in African Agro-Systems (1980-2009)

Epidemiology and Infections, **2013**, 141: 1117-1133

A21. FERGUSON KJ, CLEAVELAND S, HAYDON DT, **CARON A**, KOCK R, LEMBO T, HOPCRAFT JGC, CHARDONNET B, NYARIKI T, KEYYU J, PASTON D, KIVARIA FM

Evaluating the potential for the environmentally sustainable control of foot and mouth disease in sub-Saharan Africa

EcoHealth, **2013**, 10(3): 314-322

A20. DE GARINE-WICHATITSKY M, MIGUEL E, MUKAMURI B, GARINE-WICHATITSKY E, WENCELIUS J, PFUKENYI D, **CARON A**

Coexisting with wildlife in Transfrontier Conservation Areas in Zimbabwe: cattle owners' awareness of disease risks and perception of the role played by wildlife

Comparative Immunology and Microbiology of Infectious Diseases, **2013**, 36: 321–332

A19. DE GARINE-WICHATITSKY M, **CARON A**, KOCK R, TSCHOP R, MUNYEME M, HOFMEYR M, MICHEL A

A review on bovine tuberculosis at the wildlife/livestock/human interface in sub-Saharan Africa

Epidemiology and Infection, **2013**, 141: 1342–1356

A18. CUMMING GS, SHEPARD E, OKANGA S, **CARON A**, NDLOVU M, PETERS JL

Host associations, biogeography, and a first phylogeny of avian malaria in southern African waterfowl

Parasitology, **2013**, 140(2): 193-201.

A17. **CARON A**, MIGUEL E, GOMO C, MAKAYA P, PFUKENYI D, HOVE T, FOGGIN C, DE GARINE-WICHATITSKY M

Relationship between burden of infection in ungulate populations and wildlife/livestock interfaces

Epidemiology and Infections, **2013**, 141: 1522–1535

A16. MUNDAVA J, **CARON A**, GAIDET N, COUTO F, COUTO T, DE GARINE-WICHATITSKY M, MUNDY P

Factors influencing long-term and seasonal waterbird abundance and composition at two adjacent lakes in Zimbabwe

Ostrich, **2012**, 83(2): 69-77

A15. GOMO C, MUSARI S, DE GARINE-WICHATITSKY M, **CARON A**, PFUKENYI DM, VAN HEERDEN H

Detection of Brucella abortus in Chiredzi district in Zimbabwe

Onderstepoort Journal of Veterinary Research, **2012**, 79(1): 5

A14. GOMO C, DE GARINE-WICHATITSKY M, CARON A, PFUKENYI DM

Survey of brucellosis at the wildlife–livestock interface on the Zimbabwean side of the Great Limpopo Transfrontier Conservation Area

Tropical Animal Health & Production, **2012**, 44: 77-85

A13. GAIDET N, **CARON A**, CAPPELLE J, CUMMING GC, BALANÇA G, HAMMOUMI S, CATTOLI G, ABOLNIK C, SERVAN DE ALMEIDA R, GIL P, FEREIDOUNI SR, GROSBOIS V, TRAN A, MUNDAVA J, FOFANA B, OULD EL MAMY AB, NDLOVU M, MONDAIN-MONVAL JY, TRIPLET P, HAGEMEIJER W, KARESH WB, NEWMAN SH, DODMAN T

Understanding the ecological drivers of avian influenza virus infection in wildfowl: a continental scale study across Africa

Proceedings of the Royal Society B, 2012, 279: 1131-1141

A12. GAIDET N, OULD EL MAMY AB, CAPPELLE J, **CARON A**, CUMMING GS, GROSBOIS V, GIL P, HAMMOUMI S, DE ALMEIDA RS, FEREIDOUNI SR, CATTOLI G, ABOLNIK C, MUNDAVA J, FOFANA B, NDLOVU M, DIAWARA Y, HURTADO R, NEWMAN SH, DODMAN T, BALANCA G

Investigating Avian Influenza Infection Hotspots in Old-world Shorebirds

PLoS ONE, **2012**, 7(9): e46049

A11. **CARON A**, DE GARINE-WICHATITSKY M, NDLOVU M, CUMMING GS

Linking avian communities and avian influenza ecology in Southern Africa using epidemiological functional groups

Veterinary Research, **2012**, 43:73

A10. CUMMING GS, **CARON A**, ABOLNIK C, CATOLLI G, BRUINZEEL L, BURGER CE, CECCHETTIN K, CHIWESHE N, MOCHOTLHOANE B, MUTUMI G, NDLOVU M

The ecology of influenza A viruses in wild birds in southern Africa

EcoHealth, **2011**, 8(1): 4-13

A09. **CARON A**, ABOLNIK C, MUNDAVA J, GAIDET N, BURGER CE, MOCHOTLHOANE B, BRUINZEEL L, CHIWESHE N, DE GARINE-WICHATITSKY M, CUMMING GS

Persistence of Low Pathogenic Avian Influenza Virus in Waterfowl in an African Ecosystem

EcoHealth, **2011**, 8(1): 109-115

A08. DE GARINE-WICHATITSKY M, **CARON A**, GOMO C, FOGGIN C, DUTLOW K, PFUKENYI D, LANE E, LE BEL S, HOFMEYR M, HLOKWE T, MICHEL A

Bovine Tuberculosis in Buffaloes, Southern Africa

Emerging Infectious Diseases, **2010**, 16(5): 884-885

A07. **CARON A**, DE GARINE-WICHATITSKY M, GAIDET N, CHIWESHE N, CUMMING GS

Estimating dynamic risk factors for pathogen transmission using community-level bird census data at the wildlife/domestic interface

Ecology and Society, **2010**, 15(3): 25

A06. **CARON A**, GAIDET N, DE GARINE-WICHATITSKY M, MORAND S, CAMERON E

Evolutionary Biology, Community Ecology and Avian Influenza

Infection, Genetics and Evolution, **2009**, 9(2): 298-303

A05. GAIDET N, DODMAN T, **CARON A**, BALANÇA G, DESVAUX S, GOUTARD F, CATTOLI G, MARTIN V, TRIPODI A, LAMARQUE F, HAGEMEIJER W, MONICAT F

Influenza surveillance in wild birds in Eastern Europe, the Middle East and Africa: preliminary results from an ongoing FAO-led survey

Journal of Wildlife Diseases, **2007**, 43(3): Supplement 2007, S22-S28

A04. GAIDET N, DODMAN T, **CARON A**, BALANÇA G, DESVAUX S, GOUTARD F, CATTOLI G, LAMARQUE F, HAGEMEIJER W, MONICAT F

Avian Influenza Viruses in Water Birds, Africa

Emerging Infectious Diseases, **2007**, 13(4): 626-629

A03. ETTER E, DONADO P, JORI F, **CARON A**, GOUTARD F, ROGER F

Risk analysis and bovine tuberculosis, a re-emerging zoonosis

Annals of the New York Academy of Sciences, **2006**, 1081:)61-73.

A02. ARTOIS M, **CARON A**, LEIGHTON FA, BUNN C, VALLAT B

La faune sauvage et les maladies émergentes

Revue Scientifique et Technique de l'OIE, **2006**, 25(3): 897-912

A01. **CARON A**, CROSS PC, DU TOIT JT

Ecological implications of bovine tuberculosis in African Buffalo herds

Ecological Applications, **2003**,13(5): 1338-1345

2. Book chapters

L05. KOCK R, KOCK M, DE GARINE-WICHATITSKY M, CHARDONNET P, **CARON A**

*Livestock and buffalo (*Syncerus caffer*) interfaces in Africa: ecology of disease transmission and implications for conservation and development*

In: Melletti M, Burton J (Eds). "Ecology, Evolution and Behaviour of Wild Cattle: Implications for Conservation", 2014, Cambridge University Press, Cambridge: 431-445

L04. DE GARINE-WICHATITSKY M, FRITZ H, CHAMINUKA P, **CARON A**, GUERBOIS C, PFUKENYI D, MATEMA C, JORI F, MURWIRA A

Consequences of animals crossing the edges of Transfrontier Parks

In: Andersson J, de Garine-Wichatitsk M, Dzingirai V, Giller K and Cumming D (Eds). "Transfrontier conservation areas. People living on the edge", 2013, Earthscan, London: 137-162

L03. MURWIRA A, DE GARINE-WICHATITSKY M, ZENGEYA F, POSHIWA X, MATEMA S, **CARON A**, GUERBOIS C, HELLARD E, FRITZ H

Resource gradients and movements across the edge of transfrontier parks

In: Andersson J, de Garine-Wichatitsk M, Dzingirai V, Giller K and Cumming D (Eds). "Transfrontier conservation areas. People living on the edge", 2013, Earthscan, London: 123-136

L02. **CARON A**, MORAND S, DE GARINE WICHATITSKY M

Epidemiological Interaction at the Wildlife/Livestock/Human Interface: Can We Anticipate Emerging Infectious Diseases in Their Hotspots? A Framework for Understanding Emerging Diseases Processes in Their Hot Spots

In: Morand S, Beaudeau F, Cabaret J (Eds). "New Frontiers in Molecular Epidemiology of Infectious Diseases", 2012, Springer, Heidelberg: 311-332

L01. DUBE T, MURWIRA A, **CARON A**, DE GARINE-WICHATITSKY, M

*Preliminary results on the permeability of veterinary fences to buffalo (*Syncerus caffer*) and cattle in Gonarezhou National Park, Zimbabwe*

In: Ferguson K & Hanks J (Eds). "Fencing Impacts: A review of the environmental, social and economic impacts of game and veterinary fencing in Africa with particular reference to the Great Limpopo and Kavango-Zambezi Transfrontier Conservation Areas", 2010 Pretoria: Mammal Research Institute: 228-232

3. Peer reviewed article in Journals without an ISI IF

B10. MIGUEL E, EL IDRISSI A, CHEVALIER V, **CARON A**, FAYE B, PEIRIS M, ROGER F

Ecological and epidemiological roles of camels: lessons from existing and emerging viral infections

EMPRES - Animal Health, **2015**, in press

B09. ZENGEYA F, MURWIRA A, de GARINE-WICHATITSKY M, **CARON A**, CORNELIS D, GANDIWA P

Spatial overlap between sympatric wild and domestic herbivores links to resource gradients

Remote Sensing Applications: Society and Environment, **2015**, in press

B08. FIGUIÉ M, BINOT A, **CARON A**

Sauvage et Domestique, Homme et Animaux: une surveillance sanitaire qui brouille les frontières

Revue d'Anthropologie des Connaissances, **2015**, 9(2): 163-188

B07. PEYRE M, GAIDET N, **CARON A**, CAPPELLE J, TRAN A, ROGER F

Influenza aviaire dans le monde: situation au 31 janvier 2015

Bulletin de l'ANSES, **2015**, 67: 10-14

B06. **CARON A**, DE GARINE-WICHATITSKY M, ROGER F

Bovine tuberculosis: a double-edged issue at the human/livestock/wildlife interface in Africa

EMPRES - Animal Health, **2014**, 44(2): 10-13

B05. ZVIDZAI M, MURWIRA A, CARON A, DE GARINE-WICHATITSKY M

Waterhole use patterns at the wildlife/livestock interface in a semi-arid savanna of Southern Africa

International Journal of Development and Sustainability, **2013**, 2(2): Online ISSN: 2168-8662 – www.isdsnet.com/ijds

B04. **CARON A**, CUMMING GS, MUNDAVA J, CHIWESHE N, MUNDY P, DE GARINE-WICHATITSKY M

Report on a five-year avian influenza survey in the manyame catchment

Honeyguide, **2012**, 58(2): 113-120

B03. CHIWESHE N, **CARON A**

Monitoring birds through counting and ringing around the Manyame lakes, Zimbabwe

Honeyguide, **2012**, 58(2): 138-159

B02. BINOT A, CASTEL V, **CARON A**

L'interface faune-bétail en Afrique subsaharienne

Sécheresse, **2006**, 17(1-2): 349-361

B01. **CARON A**, KOCK R

The health issue in mixed wildlife and livestock system: the example of Rinderpest eradication in Africa

Game and Wildlife Science, **2004**, 21(4): 529-538

4. Submitted articles

AS08. MERCAT M, RUPPE E, MASSOT M, CLERMONT O, ANDREMONT A, MIGUEL F, DE GARINE-WICHATITKSY M, DENAMUR E, **CARON A**

Escherichia coli *populations sharing and antibioresistance gradient at a buffalo/cattle interface in southern Africa*

Resubmitted after major revision to Applied and Environmental Microbiology

AS07. DE GARINE-WICHATITSKY M, MIGUEL E, CORNÉLIS D, GROSBOIS V, **CARON A**

Measuring contacts between African buffalo and domestic cattle in the Great Limpopo Transfrontier Conservation areas: implications for modelling pathogen spill-over between wild and domestic hosts

Submitted to Journal of Applied Ecology

AS06. CHIGWENHESE L, MURWIRA A, ZENGEYA F, MASOCHA M, DE GARINE-WICHATITSKY M, **CARON A**

*Monitoring African Buffalo (*Syncerus caffer*) and Cattle (*Bos taurus*) movement across a damaged veterinary control fence at a Southern African wildlife/livestock interface*

Submitted to African Journal of Ecology

AS05. BORDES F, **CARON A**, DE GARINE-WICHATITSKY M, MORAND S

Rodent-borne diseases-based networks at rodent/human interfaces: transmission ecology in heterogeneous landscapes in Southeast Asia

Submitted to Ecological Applications

AS04. MIGUEL E, VALEIX M, GROSBOIS V, LOVERIDGE A, **CARON A**, STAPELKAMP B, MACDONALD D, DE GARINE-WICHATITSKY M, FRITZ H

Does predator presence modulate the risk of pathogen transmission from wild to domestic herbivores at the edge of protected areas?

Submitted to Ecological Applications

AS03. **CARON A**, CAPPELLE J, GAIDET N

Shifting paradigm in for the maintenance of IAV in Wild Birds

Submitted to Emerging Infectious Diseases

AS02. MUNDAVA J, CARON A, DE GARINE-WICHATITSKY M, ABOLNIK C, MUNDY P, GAIDET N

Role of breeding phenology and aggregation of waterfowl on avian influenza dynamics in southern Africa

Resubmitted after major revision to Ibis

AS01. DE GARINE-WICHATITSKY M, **CARON A**, MORAND S

Epidemiological Functional Groups: a framework for the identification of key host populations involved in the emergence of infectious diseases

Resubmitted to Emerging Infectious Diseases

5. Main conference presentations and posters

Oral presentations

CO62. DE GARINE-WICHATITSKY M, JOMANE L, TIVAPASI M, HOVE T, PFUKENYI D, MUKAMURI B, **CARON A**

Indigenous and scientific knowledge regarding the role of wildlife in tick-borne diseases epidemiology in interfaces areas of the Great Limpopo TFCA, Zimbabwe

ISVEE Conference XIV, 3-7 November **2015**, Mérida, Yucatán, México

CO61. DE GARINE-WICHATITSKY M, MORTON K, **CARON A**, ZIMBA M, MIGUEL E, MARIANNEAU P, CHEVALIER V

A preliminary assessment of Rift Valley Fever epidemiology in human/livestock/wildlife interfaces in Zimbabwe

ISVEE Conference XIV, 3-7 November **2015**, Mérida, Yucatán, México

CO60. ROGER R, CHEVALIER V, JORI F, THOMPSON P, **CARON A**, ETTER E

Southern Africa, an at-risk region for the peste des petits ruminants (PPR): the need of integrated modelling

ISVEE Conference XIV, 3-7 November **2015**, Mérida, Yucatán, México

CO59. **CARON A**, GAIDET N, CAPPELLE J, MIGUEL E, CORNELIS D, GROSBOIS V, DE GARINE-WICHATITKSY M

Disease ecology in multi-host systems at wildlife/livestock interfaces

ILRI Seminar, 10 June **2015**, ILRI Nairobi, Kenya

CO58. **CARON A**, DE GARINE-WICHATITKSY M, MIGUEL E, GROSBOIS V, FOGGIN C, HOFMEYR M, CORNELIS D

An investigation of social dynamics in Cape buffalo: implicaitons for disease transmission at wildlife/domestic interfaces in the GLTFCA

Savanna Science Network Meeting, 9-12 March **2015**, Skukuza, Kruger National Park

CO57. **CARON A**, CORNELIS D, MIGUEL E, GROSBOIS V, FOGGIN C, HOFMEYR M, DE GARINE-WICHATITSKY M

Buffalo movements at the wildlife/livestock interface in the GLTFCA: consequences for pathogen transmission

RP-PCP/AHEAD Conference, 12-15 March 2014, Hwange National Park, Zimbabwe

CO56. **CARON A**, CORNELIS D, MIGUEL E, GROSBOIS V, FOGGIN C, HOFMEYR M, DE GARINE-WICHATITSKY M

Buffalo movements at the wildlife/livestock interface along the Limpopo River and consequences for pathogen transmission: preliminary results from the BUCATIN project 2010-2014

Savanna Science Network Meeting, 9-13 March **2014**, Skukuza, Kruger National Park

CO55. DE GARINE-WICHATITSKY M, **CARON A,** MORAND S

Epidemiological Functional Groups: a framework for the identification of key host populations involved in emerging diseases

Savanna Science Network Meeting, 9-13 March **2014**, Skukuza, Kruger National Park

CO54. DE GARINE-WICHATITSKY M, **CARON A**, KOCK R, TSCHOP R, MUNYEME M, HOFMEYR M, MICHEL A

Bovine Tuberculosis In Wildlife In Africa: Where Is The Source Or The Sink At Wildlife/Livestock/Human Interfaces?

14th AITVM Conference, 25 – 29 August **2013**, Johannesburg, South Africa

CO53. DE GARINE-WICHATITSKY M, MWENJE E, **CARON A**, MUKAMURI B, MUNDY P, ETTER E, MUGABE PH, MURWIRA A, FRITZ H

Research platform «production and conservation in partnership », promoting collaborative applied research and postgraduate training to study wild/domestic interfaces in southern Africa

14th AITVM Conference, 25 – 29 August **2013**, Johannesburg, South Africa

CO52. GAIDET N, **CARON A**, CAPPELLE J, CUMMING G. BALANÇA G, HAMMOUMI S, CATTOLI G, ABOLNIK C, SERVAN DE ALMEIDA R, GIL P, FEREIDOUNI SR, GROSBOIS V, TRAN A , MUNDAVA J, FOFANA B, OULD EL MAMY AB, NDLOVU M, MONDAIN-MONVAL JY, TRIPLET P, HAGEMEIJER W, KARESH WB, NEWMAN SH, DODMAN T

Understanding the ecological drivers of avian influenza virus infection in wildfowl: a continental-scale study across Africa

AITVM 2013 International Conference, August **2013**, Johannesburg, South Africa

CO51. DE GARINE-WICHATITSKY M, **CARON A**, KOCK R, TSCHOP R, MUNYEME M, HOFMEYR M, MICHEL A

Bovine tuberculosis in wildlife in Africa: where is the source or the sink at wildlife/livestock/human interfaces?

Second One Health Conference in Africa, 16-19 April 2013, Arusha, Tanzania

CO50. DE GARINE-WICHATITSKY M, MIGUEL E, CORNELIS D, ZENGEYA F, GROSBOIS V, DUBOZ R, MURWIRA A, GANDIWA-ZISADZA P, **CARON A**

Measuring contacts between African buffalo and domestic cattle in the great Limpopo transfrontier conservation areas: implications for modelling bovine tuberculosis spill-over between wild and domestic hosts

Second One Health Conference in Africa, 16-19 April 2013, Arusha, Tanzania

CO49. DE GARINE-WICHATITSKY M, **CARON A**, KOCK R, TSCHOP R, MUNYEME M, HOFMEYR M, MICHEL A

Bovine tuberculosis in wildlife in Africa: where is the source or the sink at wildlife/livestock/human interfaces?

Savanna Science Network Meeting, 4-9 March **2013**, Skukuza, Kruger National Park

CO48. CORNELIS D, **CARON A**, MIGUEL E, TRAN, AL, DE GARINE-WICHATITSKY M

An investigation of association and fusion-fission patterns in African buffalo (Syncerus caffer caffer) based on GPS telemetry

Savanna Science Network Meeting, 4-9 March **2013**, Skukuza, Kruger National Park

CO47. SMITZ N, CORNELIS D, **CARON A**, DE GARINE-WICHATITSKY M, JORI F,KANAPECKAS KL, CHARDONNET P, MELLETTI M, MICHAUX J

Effect of habitat fragmentation on the genetic structure of southern African populations of African buffalo (Syncerus caffer)

Savanna Science Network Meeting, 4-9 March **2013**, Skukuza, Kruger National Park

CO46. DE GARINE-WICHATITSKY M, MIGUEL E, MUKAMURI B, GARINE-WICHATITSKY E, WENCELIUS J, PFUKENYI D, **CARON A**

Coexisting with wildlife in Transfrontier Conservation Areas in Zimbabwe: cattle owners' awareness of disease risks and perceptions of the role played by wildlife

Savanna Science Network Meeting, 4-9 March **2013**, Skukuza, Kruger National Park

CO45. DE GARINE-WICHATITSKY M, MIGUEL E, CORNELIS D, ZENGEYA F, GROSBOIS V, DUBOZ R, MURWIRA A, GANDIWA-ZISADZA P, **CARON A**

Measuring contacts between African buffalo and domestic cattle in the Great Limpopo Transfrontier Conservation Area: implications for modelling bovine tuberculosis spill-over between wild and domestic hosts

International Wildlife TB Conference, 9-12 September **2012**, Skukuza, Kruger National Park

CO44. SMITZ N, CORNELIS D, **CARON A**, DE GARINE-WICHATITSKY M, JORI F,KANAPECKAS KL, CHARDONNET P, MELLETTI M, MICHAUX J

Effect of habitat fragmentation on the genetic structure of southern African populations of African buffalo (Syncerus caffer)

86th Conference of the German Society of Mammalogy, 04-08 September **2012**, Frankfurt, Germany

CO43. DE GARINE-WICHATITSKY M, MIGUEL E, CORNELIS D, GROSBOIS V, PFUKENYI DM, MUKAMURI B, GOMO C, **CARON A**

Aires de conservation transfrontalières en Afrique australe et émergence de maladies à l'interface faune/bétail/homme

Wildlife Disease Association/European Wildlife Disease Association Conference, 23-27 July **2012**, Lyon, France

CO42. GAIDET N, **CARON A**, CAPPELLE J, CUMMING G, BALANÇA G, HAMMOUMI S, CATTOLI G , ABOLNIK C, SERVAN DE ALMEIDA R, GIL P, FEREIDOUNI SR5, GROSBOIS V, TRAN A6 , MUNDAVA J, FOFANA B, OULD EL MAMY AB, NDLOVU M, MONDAIN-MONVAL JY, TRIPLET P, HAGEMEIJER W, KARESH WB, NEWMAN SH, DODMAN T

Understanding the ecological drivers of avian influenza virus infection in wildfowl: a continental-scale study across Africa

8th International Symposium on Avian Influenza, April **2012**, University of

London,UK

CO41. MUNDAVA J, **CARON A**, GAIDET N, DE GARINE-WICHATITSKY M, MUNDY P

Breeding seasonality of wild duck species and implications for avian influenza epidemiology: a Zimbabwbean case study

30th World Veterinary Congress, 10-14th of October **2011**, Cape Town, South Africa

CO40. CARON, A.,DE GARINE-WICHATITSKY, M., MORAND, S

Ecology of Disease Transmission in Multi-host Systems

30th World Veterinary Congress, 10-14th of October **2011**, Cape Town, South Africa

CO39. **CARON A**, DE GARINE-WICHATITSKY M, NDLOVU M, CUMMING GS

Exploring the relation between avian communities and AIV ecology in Southern Africa using the epidemiological functional group concept

30th World Veterinary Congress, 10-14th of October **2011**, Cape Town, South Africa

CO38. **CARON A**, GROSBOIS V, ETTER E, DE GARINE-WICHATITSKY M

Risk of diffusion of a highly pathogenic avian influenza virus between wild and domestic avian compartments through wild birds in Zimbabwe

30th World Veterinary Congress, 10-14th of October **2011**, Cape Town, South Africa

CO37. DE GARINE-WICHATITSKY M, MIGUEL E, JORI F, HOFMEYR M, PFUKENYI D, FOGGIN C, **CARON A**

Prevalence of diseases at different wildlife/livestock interfaces in the Great Limpopo Transfrontier Conservation Area

30th World Veterinary Congress, 10-14th of October **2011**, Cape Town, South Africa

CO36. DE GARINE-WICHATITSKY M, FRITZ H, DZINGIRAI V, **CARON A**, ETTER E, MATOPE G, MURWIRA A, MUGABE P, MUKAMURI B, TITTONELL P, MWENJE E

Research Platform "Production and Conservation in Partnership": promoting collaborative applied research and postgraduate training to study wild/domestic interfaces in Southern Africain

30th World Veterinary Congress, 10-14th of October **2011**, Cape Town, South Africa

CO35. **CARON A**, MIGUEL E, JORI F, HOFMEYR M, PFUKENYI D, FOGGIN C, DE GARINE-WICHATITSKY M

Bovine tuberculosis survey in buffalo and cattle in the Great Limpopo Transrfontier Conservation Area

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CO34. **CARON A**, JORI F, THOMPSON P, PFUKENYI D, DE GARINE-WICHATITSKY M, HEATH L

CORUS FMD Vaccination trial in Zimbabwe: still preliminary results!

Final CORUS FMD Meeting, 4-5th of May **2011**, Skukuza, KNP, South Africa

CO33. **CARON A**, MIGUEL E, JORI F, HOFMEYR M, PFUKENYI D, FOGGIN C, DE GARINE-WICHATITSKY M

Prevalence of diseases at different wildlife/livestock interfaces in the Great Limpopo Transfrontier Conservation Area.

11th AHEAD-GLTFCA Working Group Meeting, 14-18 March **2011**, Kruger NP, South Africa

CO32. DE GARINE-WICHATITSKY M, MIGUEL E, CORNELIS D, GROSBOIS V, FOGGIN C, JORI F, HOFMEYR M, **CARON A**

Contacts between domestic cattle and African buffalo in the Great Liùmpopo Transfrontier Conservation Area: potential for disease spread

11th AHEAD-GLTFCA Working Group Meeting, 14-18 March **2011**, Kruger NP, South Africa

CO31. DE GARINE-WICHATITSKY M, MURWIRA A, PFUKENYI D, **CARON A**, MUNDY P, FRITZ H, MWENJE E

Activities of the Research Platform Production and Conservation in Partnership (RP-PCP) on wildlife-livestock interface in the SE lowveld of Zimbabwe: outputs after 3 years

11th AHEAD-GLTFCA Working Group Meeting, 14-18 March **2011**, Kruger NP, South Africa

CO30. DE GARINE-WICHATITSKY M, MIGUEL E, CORNÉLIS D, GROBOIS V, FOGGIN C, JORI F, HOFMEYR M, **CARON A**

Contacts between domestic cattle and African buffalo in the Great Limpopo Transfrontier Conservation Area: potential for disease spread

Savanna Science Network Meeting 2-4 March, **2011**, Mopane Camp, Kruger National Park

CO29. **CARON A**, MIGUEL E, JORI F, HOFMEYR M, PFUKEYNI D, FOGGIN C, DE GARINE-WICHATITSKY M

Prevalence of diseases at different wildlife/livestock interfaces in the Great Limpopo Transfrontier Conservation Area

Savanna Science Network Meeting 2-4 March, **2011**, Mopane Camp, Kruger National Park

CO28. **CARON A,** DE GARINE-WICHATITSKY M, GAIDET N, ABOLNIK C, CUMMING GS

Estimating and validating a dynamic risk factor model for pathogen transmission using community-level bird census data : avian influenza at the waterfowl/domestic bird interface in Zimbabwe

EcoHealth, 7(suppl.): 164 (p.S121) International One Health Congress, 14-16 February **2011**, Victoria, Australia

CO27. **CARON A**, DE GARINE-WICHATITSKY M, GOMO C, FOGGIN C, MIGUEL E

Emergence of bovine tuberculosis in wildlife in Southern Africa : a threat for livestock

EcoHealth, 7(suppl.): 161 (p.S66) International One Health Congress, 14-16 February **2011**, Victoria, Australia

CO26. **CARON A**, DE GARINE-WICHATITSKY M, MORAND S

Parasite community ecology and epidemiological interactions at the wildlife/domestic/human interface : can we anticipate emerging infectious diseases in their hotspots?

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CO25. JORI F, **CARON A**, THOMPSON P, PFUKENYI, D, DE GARINE-WICHATITSKY M, HEATH L

Possible evidence for silent circulation of a SAT 3 FMD strain in cattle populations adjacent of the Gonarezhou National Park, Zimbabwe

SASVEPM meeting, August **2010**, South Africa

CO24. **CARON A**, MORAND S, DE GARINE-WICHATITSKY M

Using the community of pathogens to infer interspecific epidemiological interactions at the wildlife-domestic interface: a tool for exploring emerging diseases's processes in their hotsposts

10th AHEAD meeting, 24-27 February **2010**, Hazyview, South Africa

CO23. DE GARINE-WICHATITSKY M, **CARON, A**, MURWIRA A, ZENGEYA F, ZVIDZAI M, DUBE T, GOMO C, PFUKENYI D, ZISADZA P

Activities of the Research Platform Production and Conservation in Partnership (RP-PCP) on wildlife-livestock interface in the SE lowveld of Zimbabwe: an overview and updates on disease prevalence and contacts between wild and domestic ungulates

10th AHEAD meeting, 24-27 February **2010**, Hazyview, South Africa

CO22. GEOGHEGAN C, LOVEMORE M, **CARON A**, CUMMING D, GETZ W, DE GARINE-

WICHATITSKY M, ROBERTSON M, CAMERON E

Pathogens, Parks and People: Assessing the Role of Disease in Trans-Frontier Conservation Area Development

10th AHEAD meeting, 24-27 February **2010**, Hazyview, South Africa

CO21. **CARON A**, HEATH L, PFUKENYI D, DE GARINE-WICHATITSKY M, THOMSON P, JORI F

Evaluation of vaccination efficiency against FMD and virus circulation in cattle populations at the wildlife/livestock interface within the Zimbabwean lowveld

CORUS Meeting, 22-23 February **2010**, Hazyview, South Africa

CO20. **CARON A**, CUMMING GS, ABOLNIK C, CHIWESHE N, DE GARINE-WICHATITSKY M

A community approach to explore host-pathogen interactionsat the wildlife/domestic interface: Avian Communities and Avian Influenza in a Zimbabwean Ecosystem

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CO19. **CARON A**, CUMMING GS, ALBONIK C, CHIWESHE N, DE GARINE-WICHATITSKY M

A community approach to explore host-pathogen interactions at the wildlife/domestic interface: avian communities and avian influenza in a Zimbabwean ecosystem

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CO18. DE GARINE-WICHATITSKY M, GOMO C, JOMANE L, PFUKENYI D, HOVE T, FOGGIN C, **CARON A**

Prevalence of main diseases in wild and domestic ungulates at three contrasted wildlife/livestock interfaces in the South East Lowveld of Zimbabwe

ISVEE Conference XII, 10-14 August **2009**, Durban, South Africa

CO17. **CARON A**

Introductive note

"Host-Pathogen Community Interaction: from Mobility to Contact" ATP First Emergence Meeting, 28-29 avril **2009**, Montpellier, France.

CO16. DE GARINE-WICHATITSKY M, **CARON A**

Measuring contact rates between wild and domestic ungulates to estimate pathogen transmission

"Host-Pathogen Community Interaction: from Mobility to Contact" ATP Emergence First Meeting, 28-29 avril **2009**, Montpellier, France

CO15. **CARON A**, DE GARINE-WICHATITSKY M

Using community ecology to infer epidemiological interactions.

"Host-Pathogen Community Interaction: from Mobility to Contact" ATP Emergence First Meeting, 28-29 avril **2009**, Montpellier, France

CO14. GAIDET N, CATTOLI G, DODMAN T, HAMMOUMI S, **CARON A**, CAPPELLE J, MONNE I, FUSARO A, MANU S, HAGEMEIJER W, TAKEKAWA JY, NEWMAN SH

Epidemiological investigations of Avian Influenza Viruses in wild birds in Eastern Europe, the Middle East and Africa

Second Pan-European Duck Symposium, 23-25 March **2009**, Arles, France

CO13. **CARON A**, GOMO C, JOMANE L, PFUKENYI D, FOGGIN C, HOVE T, DE GARINE-WICHATITSKY M

Wildlife-livestock interface in the SE lowveld of Zimbabwe: first results on disease prevalence in wild and domestic ungulates

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CO12. DE GARINE-WICHATITSKY M, ZENGEYA F, ZVIDZAI M, MURWIRA A, ZISADZA P, **CARON A**

Wildlife-livestock interface in the SE lowveld of Zimbabwe: first results on distribution and contacts between wild and domestic ungulates

9th AHEAD meeting, 4-6 March **2009**, Namaacha, Mozambique

CO11. CHEVALIER V, **CARON A**, OLIVE MM, REYNES JM, DE GARINE-WICHATITSKY M

Impact of the wildlife on Rift Valley fever epidemiology.

OIE Séminaire Régional pour le control et la lutte contre la Fièvre de la Vallée du Rift en Afrique Australe, 16-18 Fevrier **2009**, Bloemfontein, South Africa

CO10. **CARON A,** FRITZ H, MORAND S, DE GARINE-WICHATITSKY M

Network of Interaction, Geographic Mosaic of Coevolution and Community Ecology: Sanitary Risk at the Wildlife / Livestock Interface

Meeting of the two GDRIs on Biodiversity and Sustainable Use in the context of Global Change - Madagascar and Southern Africa at the Corum and CNRS, 3rd November **2008**, Montpellier, France

CO09. GAIDET N, DODMAN T, HAMMOUMI S, CATTOLI G, CAPPELLE J, **CARON A**, HAGEMEIJER W, GIL P, FEREIDOUNI S, ADENIYI T, MULLER C, TAKEKAWA J, KARESH W, MONICAT F, NEWMAN SH

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CO08. DE GARINE-WICHATITSKY M, **CARON A**

Update on CIRAD project in the Zimbabwean South East Lowveld

8th AHEAD meeting, 5-7 Mars **2008**, Nelspruit, South Africa.

CO07. GAIDET N, DODMAN T, GIL P, CAPPELLE J, HAMMOUMI S, CATTOLI G, **CARON A**, TERREGINO C, SALOMONI A, HAGEMEIJER W, TAKEKAWA JY, MONICAT F, LUBROTH J, DOMENECH J, NEWMAN SH

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CO06. GOUTARD F, DESVAUX S, ROGER F, GAIDET N, **CARON A**, BALANÇA G, MONICAT F, DIESEL G, WIELAND B, GUITAN J, COSTARD S, PFEIFFER D, HAGEMEIJER W, LEMENACH A, VONDOBSCHUETZ S, TRIPODI A, MARTIN V

Regional training workshops for early detection and prevention of avian influenza

12th International Conference of the Association of Institutions for tropical Veterinary Medicine, August **2007**, Montpellier, France

CO05. MARTIN V, DESIMONE L, LEMENACH A, NEWMAN SH, ROGER F, GOUTARD F, WIELAND B, GUITIAN J, SOARES-MAGALHAES R, **CARON A**, GAIDET N, PFEIFFER D

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CO04. **CARON A**

CIRAD Lowveld Livestock Project. *7th AHEAD meeting*

8-9 Mars **2007**, Pequenos Libombos Dam, Mozambique

CO03. GAIDET N, DODMAN T, **CARON A**, BALANÇA G, DESVAUX S, CATTOLI G, MARTIN V, TRIPODI A, HAGEMEIJER W, MONICAT F

Surveillance of wild birds in Africa, ongoing FAO-led studies

FAO/OIE International Scientific Conference on Avian Influenza and Wild Birds, May **2006**, Roma, Italy

CO02. **CARON A,** KOCK R

The health issue in mixed wildlife and livestock systems: the example of Rinderpest eradication in Africa

6th International Wildlife Ranching Symposium, July **2004**, Paris, France

CO01. **CARON A**, CROSS P, DU TOIT JT

Impact of Bovine Tuberculosis on African Buffalo in Kruger National Park

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First Price Student Award

Poster presentations

PO16. ZISHIRI S, MUKAMURI B, **CARON A**, DE GARINE-WICHATITSKY M

Effect of improved macro-economic regime on small-scale farmers' capacity to access and manage cattle in Sengwe Communal Land, Zimbabwe"

14th AITVM Conference, 25 – 29 August **2013**, Johannesburg, South Africa.

PO15. DE GARINE-WICHATITSKY M, MWENJE E, MANJENGWA J et al., **CARON A**

Research platform «Production and Conservation in Partnership », promoting collaborative applied research and postgraduate training to study wild/domestic interfaces in southern Africa

Savanna Science Network Meeting, 4-9 March **2013**, Skukuza, Kruger National Park. Poster

PO14. SMITZ N, CORNELIS D, **CARON A**, DE GARINE-WICHATITSKY M, JORI F, KANAPECKAS KL, CHARDONNET P, MELLETTI M, MICHAUX J

Effect of habitat loss on austral African populations of Cape buffalo (Syncerus caffer caffer) based on microsatellite analysis

86th Annual Conference of the German Society of Mammalogy, 4-8 September **2012**, Frankfurt, Germany

PO13. MIGUEL E, GROSBOIS V, **CARON A**, BOULINIER T, CORNELIS D, FRITZ H, JORI F, DE GARINE-WICHATITSKY M

Wildlife-livestock interactions: frequency of contact and incidence of foot-and-mouth disease in cattle populations at the periphery of protected areas in southern Africa

Conference on FMD, OIE/FAO, June **2012**, Bangkok, Vietnam

PO12. JORI F, **CARON A**, THOMPSON PN, DWARKA R, FOGGIN C, DE GARINE-WICHATITSKY M, HOFMEYR M, VAN HEERDEN J, HEATH L

Monitoring foot and mouth disease virus dynamics within the Great Limpopo Transfrontier Conservation Area (GLTFCA)

Conference on FMD, OIE/FAO, June **2012**, Bangkok, Vietnam

PO11. **CARON A**, ETTER E, JORI F, PFUKENYI D, NJAGU C, MURWIRA A, MIGUEL E, DE GARINE-WICHATITSKY M

Environmental and ecological drivers of Foot-and-Mouth disease outbreaks at the wildlife/livestock interface in the GLTFCA

Savanna Science Network Meeting, 14-18 of March 2011, KNP, South Africa.

PO10. **CARON A**, DE GARINE-WICHATITSKY M, GAIDET N, CHIWESHE N, CUMMING GS

Estimating dymanic risk factors for pathogen transmission using community-level bird census data: can we follow pathogen movements?

Premier Colloque national d'écologie scientifique, September **2010**, Montpellier, France

PO09. **CARON A**, DE GARINE-WICHATITSKY M, MORAND S

Parasite community ecology and epidemiological interactions at the wildlife/domestic/human interface

Premier Colloque national d'écologie scientifique, September **2010**, Montpellier, France

PO08. **CARON A**, HEATH L, PFUKENYI D, DE GARINE-WICHATITSKY M, JORI F

FMD vaccination trial and viral circulation at a wildlife/livestock interface in the South-East Lowveld of Zimbabwe.

FMD International Symposium and Workshop, 12-14th of April **2010**, Melbourne, Australia

PO07. **CARON A**, PFUKENYI D, MURWIRA A, NJAGU C, DE GARINE-WICHATITSKY M

Seasonal and spatial patterns of Foot-and-Mouth Disease outbreaks in domestic cattle at the wildlife/livestock interface in Zimbabwe with particular attention to the South East Lowveld (1997-2006)

FMD International Symposium and Workshop, 12-14th of April **2010**, Melbourne, Australia

P006. DE GARINE-WICHATITSKY M, PFUKENYI D, ZISADZA P, BODY G, MAKWANGUDZE J, **CARON A**

Seroprevalence of Foot-and-Mouth disease at the wildlife/livestock interface in the South-east Lowveld of Zimbabwe: influence of home range overlap

FMD International Symposium and Workshop, 12-14th of April **2010**, Melbourne, Australia

PO05. ZENGEYA F, MURWIRA A, **CARON A,** DE GARINE-WICHATITSKY M

Rangeland utilization within the home range of cattle, Malipati Communal Land, Zimbabwe

10th AHEAD meeting, 24-26 February **2010**, Haziview, South Africa.

PO04. GOMO C, DE GARINE-WICHATITSKY M, **CARON A**, PFUKENYI D

A survey of Brucellosis in wild and domestic animals in the South-East Lowveld of Zimbabwe

10th AHEAD meeting, 24-26 February **2010**, Haziview, South Africa.

PO03. MUNDAVA J, MUNDY P, **CARON A**, GAIDET N, COUTO T, COUTO F, DE GARINE-WICHATITSKY M

An ecological approach to estimating the risks of introduction, maintenance and spread of avian influenza viruses in the chivero and manyame ecosystem.

12th Conference of the International Society for Veterinary Epidemiology and Economics (ISVEE XII), August **2009**, Durban, South Africa

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PO02. MUNDAVA J, MUNDY P, CUMMING G, **CARON A**, GAIDET N, COUTO T, COUTO FM, DE GARINE-WICHATITSKY M

Seasonal trends in waterbird numbers, community structure and species diversity in Lakes Chivero and Manyame (Zimbabwe), 1993-2003.

Second Pan-European Duck Symposium, 23-25 March **2009**, Montpellier, France

PO01. DE LA ROCQUE S, ROGER F, **CARON A**

Changes and vectorial emerging diseases: original approaches and extended partnership to face a serious risk

World Veterinary Congress, **2002**, Tunis, Tunisia & Regards Croisés sur les changements globaux, Arles, France

VI. Research Project

"The ecology of infectious disease transmission in multi-host systems within African socio-ecosystems"

A. Preamble

Who am I? Where am I going? Scientifically speaking, these are the two questions that need to be answered in this HDR thesis.

I was far from being born a researcher or an academic. However, I knew quite early that I wanted to work at the interface between development and conservation in developing countries, preferentially in Africa, the phantasmagorical land for young Europeans dreaming about adventures "in the wild" (at least at the end of the XXth century).

Involved for a few years (2002-2005) in voluntary or professional work on wildlife, wildlife diseases and diseases transmitted at wildlife/livestock interfaces in Africa, I quickly realised that Cirad was an institution where I could do what I wanted to do. The decision to do whatever possible to get a position at Cirad was what dragged me into academic life, not an objective *per se* initially. Around 2005, the "Science au Centre" policy of Cirad, trying to insufflate more academic research into the largely development-based approach implemented until then, instated that any new recruit would need to have a PhD. I accepted the challenge after some discussions with Cirad colleagues and friends.

This "figure imposée", the PhD thesis, initiated the beginning of a professional transformation that drastically changed the way I work on a daily basis, I see my career and I envisage how research and development can in collaboration contribute to promote a better coexistence between People and Nature. Ten years down the line, I see myself as a researcher, primarily, with some gaps of knowledge due to my late conversion but maybe with an advantage of not coming from the standard mould.

I arrived in Zimbabwe in April 2006 for Cirad to start my PhD and left the country in January 2015, with a full-time researcher position obtained in January 2010. The end of this chapter of almost 9 years of my professional life is also an argument to submit my HDR now, during a transition phase when I can wrap-up what I have been doing and think about what lays ahead (and have a bit of time to think and write about it in between projects' cycles).

I see the HDR, this typically French degree with no interference at all with the external scientific world where I mainly exist, as an excellent exercise to take the time to think about my position as a researcher within my fields of interest and my research environment, including international and national collaborators and Cirad colleagues. I think I have a clear understanding of where I am, where I am going

and with whom I should go there and hope that, by the end of this exercise, this perception will be comforted.

So let's engage on this stopover on my own scientific voyage (I tend to see myself on that one-palm-tree small island of cartoons), to look backward and forward as clearly as possible and follow this thin red line that drives my research interest and motivation.

B. Global Context

1. Sustainable coexistence between Man & Nature?

Within the dogma of Darwinian theory (Darwin 1859) - meaning excluding from the start any conception of the human species as special and/or "elected" - , one can observe that the human species is currently impacting the earth - so far the only place in the universe where it can survive - to an extent that not only threatens local and global biological processes but also potentially compromise its own survival (Palumbi 2001; Sachs et al. 2009). Recently, Humanity has come to realise that biodiversity conservation is a necessary variable in its equation for life on earth and that a compromise with human development and its exploitation of natural resources needs to be found globally (Adams et al. 2004). This coexistence between Man & Nature (here defined as species diversity and biological processes) is a worldwide necessity, meaning that it applies everywhere and for everyone. Humans are one component of Nature and Nature is a public good for Humanity that needs to be protected for the future.

Economic richness and development levels are not homogeneously distributed across human populations. Reasons for these heterogeneities result from environmental constraints, the heterogeneity in the initial distribution of natural resources (including potentially domesticable plant and animal species) and social and historical contingencies of human societies (Diamond 1997; Diamond 2002; Diamond 2005; Morris 2011; Ferguson 2012). As a consequence, some regions of the world are much less developed (technically, economically) than others. These so-called developing countries often rely on natural resource extraction and lack proper basic state services such as education for example. These inequalities have been linked to environmental degradation in concepts such as the "environmental Kuznets curve" which stipulates (with some controversies) that environmental degradation and human development are related by a "U-shaped" curve indicating that as development increases, the impact of human societies on the environment decreases (Boyce 1994; Stern, Common & Barbier 1996). In less developed countries, with a high impact on the environment, the level of awareness on the link between Human and Nature has not yet fully emerged as a societal challenge because crucial development steps need to be achieved before (notably in the sphere of education). There, the ecological and technical transitions still pending will therefore have to be achieved within the limits of what Nature can "take" without compromising ecological processes (Tittonell 2013). If the type of relationship between economic development and environment degradation is still debated, increased level of education and better livelihoods can surely help locally the development of conservation conscientiousness and the decrease in environmental degradation.

Within these developing countries, many of the biodiversity hotspots where protected areas have been gazetted are located in the very regions where the poorest and most marginalised human communities live (Andersson & Cumming 2013; Caron et al. 2013; Dzingirai et al. 2013). There, a combination of poor livelihoods, poor levels of education and other government services disconnect completely human populations' aspirations targeted by necessity at extracting as much resources from the environment as they can to reach food security and

environmental conservation needs to achieve sustainability. The link between natural resource use and poverty is not always straightforward and must also be reflected in its historical, political and social dimensions (Duffy et al. 2015). In these areas, making Human and Nature sustainably coexist is a challenge, addressing important issues both for development and conservation.

The ambivalence of this challenge has not always been taken into consideration. Historically, the conservation concept, emanating from Northern (and developed) countries during the XXth century, was mainly aiming at conserving biodiversity in protected areas later called "fortresses", in complete isolation from their surroundings, especially from peripheral human communities and their activities. Progressively, this concept has evolved and the new paradigm in conservation currently percolating across the world is that of "Nature and People", recognizing the dynamic relationships between Man and Nature and embedding protected areas within the socio-ecological systems where they occur (Mace 2015).

My work fits within this paradigm. Conservation cannot be achieved without granting the human populations the most exposed to the negative impacts of conservation (e.g. human/wildlife conflicts) with decent livelihoods and opportunities for the future. Moreover, the main and first step of any conservation activity should be to secure the livelihoods of surrounding communities, if possible through the sustainable use of natural resources. I particularly focus on socio-ecosystems in developing countries where human populations' needs to make a living and the pressure on natural resources are the greatest. These socio-ecosystems can be close to urban centres or in agricultural settings. Up to now, I have mainly worked where land-use patterns were delineating more or less clearly the boundary between natural areas (e.g. protected areas, wetlands) and anthropo-systems such as agricultural land or farms.

2. Conservation policy based on protected areas creates interfaces

Mace (2015) synthetized the history of the rise of biodiversity conservation consciousness/ideology and its practical implications in terms of how conservation is implemented locally. The basis of biodiversity conservation has been and still is the protected area (PA). PAs are land-use types that limit human activities within its boundaries to some extents, from complete exclusion in national parks (except for tourism) to allowing some natural resource gathering or cultural events such as in nature reserves for example.

As mentioned previously, the creation of PAs in (southern) Africa was initially the will of Northern colonial powers during the 20th century. They were often located in arid and semi-arid ecosystems and perceived as having little agricultural value. They were chosen mainly because resource-rich ecosystems were already being exploited for agriculture and were not anymore suitable for conservation. These arid and semi-arid areas consequently have been largely neglected from major development initiatives implemented by remote political powers (including after independence) based in resource-rich areas. For diverse political and historical reasons, some human communities continue to live in these less productive arid and semi-arid ecosystems. As a result, people and protected areas today share resource-poor landscapes, often close to international borders and on the periphery of richer national centres (Caron et al. 2013).

This juxtaposition of conservation areas and communal land creates what are called "interfaces". The type of interface defines the type of interactions that can exist between land-use and their actors whether they are human, animals or plants. Often an interface refers to the actors that are in interaction across it: wildlife/livestock interface or human/nature interface (e.g. (Kock 2005).

The interface between a protected area and a communal land can exist in different physical states: the simplest type of interface is a fence separating land-use types. They are many types of fence (e.g. single vs. multiple strand, double-fence) and their efficiency and impact has been reviewed recently (Ferguson & Hanks 2010; Pfeifer et al. 2014; Woodroffe, Hedges & Durant 2014). The main purpose of fences is too segregate the movement of specific actors between land-use types, preventing the use of one side of the fence by actors from the other side. Fences are often expensive to build but also expensive to maintain especially when mega-herbivores range on one side: no fence completely resist to elephants for example.

An interface can also exist under the form of an area separating protected and production areas, often called a buffer zone. Buffer zones do not impede the movement of actors. However, the restricted activities within the buffer zone compel actors to stay on "their" side. For example, buffer zones are often hunting areas where some wildlife species are professionally hunted for commercial purposes decreasing the likelihood that wildlife will cross the buffer zone and enter communal land. Human activities are also limited in buffer zones, usually to the sustainable use of some natural resources (e.g. wood, insects, pasture), decreasing the impact of human activities in the core protected area.

Finally, interfaces can be invisible land-use boundary only drawn on maps. For example in Zimbabwe, the boundary between Hwange national park and the forestry commission forest or neighbouring communal lands is invisible *in situ*. The types of interfaces exist along a gradient ranging from "hard" boundaries or interfaces to more permeable or soft or "open" interfaces. When the interface does not refer directly to a protected area but to the wildlife and human actors, it can also be defined as a "diffuse" interface. For example, the human/rodent interface in city can occur in the streets, inside buildings and almost everywhere in the city.

The type of interface is a result of national conservation and development policies and the level of management dedicated to it. It can also have its roots in history and within very different realms such as disease surveillance (Andersson & Cumming 2013) and central power control over dominated ethnic groups (Figuié, Binot & Caron 2015). Therefore wildlife/human interfaces and the implied wildlife/livestock and wildlife/livestock/human interfaces are complex physical entities linking conservation and development, particularly in Africa. Interfaces should not just represent a separation between land-use types defining the limits where stakeholders operate. Interfaces should be the focus of research and development actions in order to foster the sustainable coexistence between conservation and development. Their management should engage all stakeholders active in both conservation and development. An “interface science” should emerge with its "interface experts" to tackle the specific and multidisciplinary issues that they raise.

3. Interfaces create interactions in complex multi-actor systems

Interfaces that link land-use types and their actors promote interactions between these actors. These interactions often influence the success or failure of conservation and development objectives (Binot, Castel & Caron 2006). These interactions can be seen as positive and negative. Negative interactions are defined as interactions that result in a negative effect on either or both side of the interface. Human/wildlife conflicts are considered as negative interactions: they can take the form of crop raiding by elephants, livestock predation by lion or human death due to encounters with dangerous wildlife (Hoare 1999; Chardonnet et al. 2010; Guerbois, Chapanda & Fritz 2012) but also disease transmission both ways at the human/livestock/wildlife interface that can threaten wildlife conservation, livestock production and human health (Kock 2005; Kock et al. 2014). Positive interactions result in the benefit of some actors without any negative impact on other actors (i.e. at the population, community or ecosystem level - not individual level). The sustainable use of natural resources is a way of "using" natural resources such as worms, honey or wildlife individuals that can benefit surrounding human populations without affecting the survival of wildlife populations and ecological processes. In most cases, tourism can be seen as a positive non-consumptive interaction. Professional or subsistence hunting can also be a positive interaction if the extraction pressure does not harm in the long run wild populations (Crosmary et al. 2013). Positive interactions can also take the form of ecosystem services when protected areas provide services to communal land (Cumming et al. 2014) such as pollination by insects or dilution effect for public or domestic animal health (Keesing & Ostfeld 2015).

In a recent book co-edited by RP-PCP members (Andersson et al. 2013), I have contributed to two chapters dealing with interactions of actors at the interface. In the first chapter (Murwira et al. 2013), we looked at resource gradients and wild and domestic animal movements across the edge of the Gonarezhou National Park in Zimbabwe. In the second chapter (de Garine-Wichatitksy et al. 2013b), we investigated about the consequences of this gradient of resources and resulting quasi-percolation process across the interface of domestic animal and people towards the national park, of wildlife sometimes attracted by resources outside the national park and of pathogens benefiting from these animal movements to move across land-use types and hosts. These gradients of resources, actors' movements result therefore in interactions, positive and negative.

Human/Nature interfaces are therefore characterised by a set of positive and negative interactions between actors, the result of which define their global state, contributing positively or negatively to the success of the coexistence between Man & Nature. A sustainable coexistence between Man & Nature requires mitigating negative interactions and promoting positive ones at human/livestock/wildlife interfaces. This systemic vision of Human/Nature interfaces requires an ecosystem-based approach. As these interfaces are characterised by interactions between anthropological and ecological systems (understanding that an anthropological system is also an ecological system), one can refer to socio-ecological ecosystems (SES)(Ostrom 2009) to highlight the complexity of the system at stake and the necessity of multidisciplinary approaches to tackle these issues. SES literature has been flourishing recently and new concepts and empirical studies keep being published. One interesting field of studies to characterise the state of interfaces

and more globally of whole ecosystems or SES has been defined as "Ecosystem health". "A healthy ecosystem is defined as being 'stable and sustainable'; maintaining its organization and autonomy over time and its resilience to stress" according to (Rapport, Costanza & McMichael 1998). This definition applies well to SES but raises interesting questions about the indicators that can be used to assess the state of the ecosystem. This systemic approach of Human/Nature interfaces to promote their coexistence is necessary.

For example, improving the health of domestic ruminants in communities in the periphery of a protected area is often seen as way of securing livelihoods of small-scale farmers and achieving food security at local level. Improving diptank control implementation to better control tick infestation and tick-born related diseases should decrease livestock mortality and/or increase fertility, leading to more heads of livestock being produced locally. The average herd size should increase in the area, a relative success for an animal health & production specialist. However the consequences of this "success" could be very different depending on the local context. First, if livestock is kept in communities mainly for showing one's social status, then the increase in livestock density can increase the pressure on the scarce natural resources in the area, such as pasture and water. The carrying capacity in the communal land can be overtaken, leading to negative consequences on the grass community, available resources and overall land degradation. In reaction, farmers could be tempted to push their herds into the protected area to access additional or better resources. This type of behaviour should increase the interaction between wild and domestic ruminants leading to more competition for resources, increased disease transmission at the livestock/wildlife interface and/or increase human/wildlife conflicts such as livestock predation. Overall, this would mean negative interactions at the interface and a negative impact on ecosystem health. Alternatively, if presented with opportunities to commercialise locally or regionally their livestock, herders could decide to destock (or not if the social context is perceived as more important). They could do so when market prices are optimum; or before the dry season, selling animals that could not survive until the next rainy season; or when they need cash (for school fees or Christmas expenses). The decrease in cattle density should decrease negative interactions at the wildlife/livestock/human interface and therefore be positive for ecosystem health.

This example shows that there is no technical silver bullet for sustainable conservation and development without a comprehensive approach of SES. Multiple thematic fields and expertise need to complement each other: ecology (functional, behavioural, community, disease), anthropology, ethnobotany, epidemiology, geography, agronomy, economy and many others. Multidisciplinary is therefore a prerequisite and inter- and trans-disciplinarity an added-value if possible (Min, Allen-Scott & Buntain 2013). In addition, multi-stakeholder interactions are necessary, including early-consultation of community members and representatives, regular exchange with decision makers and government technical services as well as scientists. The co-construction of sustainable scenario for the coexistence of Man & Nature between all stakeholders can ensure that the possible outcome of the action is understood and acknowledged by everyone (Bousquet, Mathevet & Le Page 2010; Ruankaew et al. 2010).

4. The Research Platform "Production & Conservation in Partnership"

The example of the Research Platform “Production and Conservation in Partnership” (RP-PCP, http://www.rp-pcp.org) in which I have been involved since its very inception (October 2005, Hwange meeting) illustrates the type of global approach necessary to promote the coexistence of Human & Nature in SES.

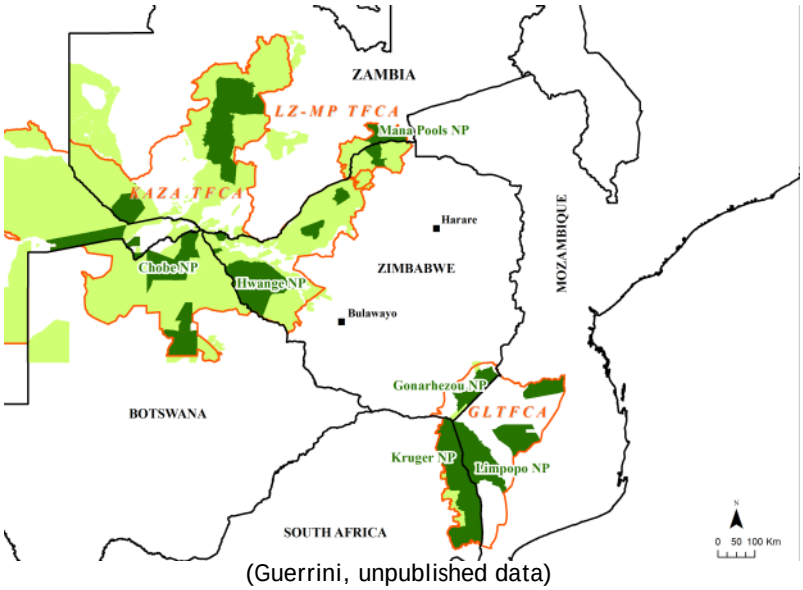
The RP-PCP was established in 2007, formally associating the University of Zimbabwe and the National University of Science and Technology with two French research organisations, Cirad (Agricultural Research for Development) and CNRS (French National Research Centre). Cirad has been conducting applied agricultural research in Zimbabwe since the beginning of the 90s and the maturity of its relationship with Zimbabwean research partners made the creation of this platform possible.

The overall objective of the RP-PCP is to contribute to sustainable development, biodiversity conservation and improved rural livelihoods in Southern Africa, through strengthening national research capacities, multidisciplinary approaches and institutional partnerships with a focus on protected areas and neighbouring production. The RP-PCP seeks to promote applied research on wild-domestic interfaces in order to address issues related to the coexistence of Man and Nature, by mitigating development and conservation activities. This means mitigating negative interactions such as human-elephant conflict, livestock predation by wild carnivores or disease transmission between wild and domestic ungulates and promoting opportunities such as natural resource management including wood, worms or wild species harvesting. This research framework seeks to understand the links between heterogeneity and sustainability of SES in the context of wild-domestic interfaces.

The RP-PCP functions thanks to an Advisory Board and a Steering Committee transformed into a Scientific Committee in 2015. Externally funded projects (e.g. European Union, French National Research Agency - ANR, French Ministry of Foreign Affairs through the French Embassy in Zimbabwe, FAO) develop research and development-based projects. Research is mainly implemented through post-graduate student projects (MSc, MPhil or PhD). Staff involved in the activities of the RP-PCP in 2015 comprises an international multidisciplinary group of more than 70 senior researchers, academics and post-graduate students from a wide range of disciplines, including Social Sciences, Agronomy, Ecology, Geography and Veterinary Sciences to cite a few. This multidisciplinary group is organized into four broad thematic areas: animal health and environment, functional ecology at wildlife/livestock/human interfaces, conservation and agriculture, and natural resources governance and institutions.

The transfrontier conservation areas (TFCAs) of southern Africa are complex SES in which traditional agricultural activities share savannah habitat with African wildlife. For farming systems that are already fragile as a result of climatic, economic and political constraints, the interactions between Man and Nature are often fraught with conflict (crop destruction by elephants, or poaching). The platform has selected 3 TFCAs as its priority areas: Gonarezhou national park (NP) in the GLTFCA, Hwange NP in the KAZA-TFCA and the mid-Zambezi valley/Lower Zambezi-Mana Pools TFCA (Figure 2).

Figure 2: Map of the 3 TFCAs targeted by the RP-PCP

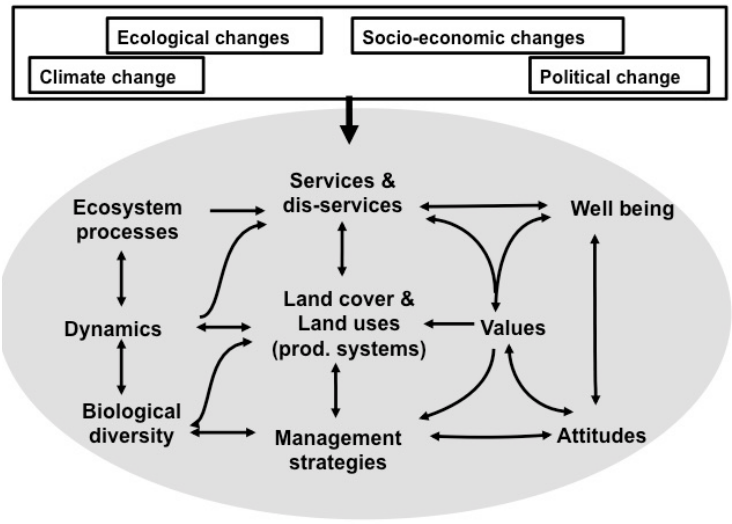


(Guerrini, unpublished data)

Since its inception, the RP-PCP has aimed to promote applied scientific studies by providing support to post-graduate students, the vast majority of them being Zimbabwean. So far, around 30 postgraduate students have completed their thesis (MPhil, MSc or PhD) and 38 are actively under RP-PCP supervision, including 45 Zimbabweans, 16 Europeans and 10 SADC students. All together, they have produced 66 research articles, 17 book chapters and numerous (>100) communications during international conferences and regional workshops. Some technical and expertise reports have also been produced.

As a group of multidisciplinary researchers focusing on the same overall objective, the RP-PCP members have for the last 5 years tried to develop an overall framework for their research in the context of the RP-PCP. The latest of this framework is displayed in Figure 3. It does not constitute a final version and I will not enter into the underlying explanations and hypotheses but will mention that we (the RP-PCP group) intend to finalise it with the help of postgraduate students trying to "map" the RP-PCP activities on this framework in order to identify potential gaps in research and reframe it if necessary.

Figure 3: RP-PCP Research Framework



Presented by M. de Garine-Wichatitsky during the last RP-PCP Advisory Board in December 2014, adapted from (Guerbois, Chapanda & Fritz 2012).

This research approach requires multidisciplinary collaborations involving "specialists" in their own fields, implementing applied and thematic research, often in collaboration with other specialists from other thematic fields. However the global approach aiming at promoting the coexistence of Human & Nature in these contexts requires also what I will call "generalists", defined here as researchers interested in the big picture, promoting the synthesis of the research produced in each thematic fields. This is not an easy task and it requires transcending one's own speciality and immersing oneself in the turbid waters of new disciplines.

My research agenda has been deeply impacted by my experience in the RP-PCP. First as a PhD student, I observed the creation of this entity and how it matured progressively into something larger than its constituent parts. In 2012, I was appointed as the co-coordinator of the platform, seconding the coordinator and getting more and more exposed to the functioning of the platform and the global research objective of it. In 2015, I became the secretary coordinator of the RP-PCP and the "animateur du Dispositif Prioritaire" for Cirad. Today, I cannot envisage my research independently from a bigger picture, the blue print of which has been laid before me by the RP-PCP in the recent years. If my speciality is the title of this HDR thesis, I always keep in mind that it fits into the global framework of the coexistence between Man & Nature in SES. Therefore, in addition, I try to be a generalist and to contribute to the overall agenda by investing some time in the scientific animation of the RP-PCP. This generalist approach can be found in a number of co-authored publications (Binot, Castel & Caron 2006; de Garine-Wichatitksy et al. 2013b; Ferguson et al. 2013; Murwira et al. 2013; Cumming et al. 2015b; Figuié, Binot & Caron 2015).

5. Health as a public good in SES

Within the context of SES as presented above, "health" is an interesting entry point. The World Health Organisation (WHO) defines in 1946 health as "a state of

complete physical, mental, and social well-being and not merely the absence of disease or infirmity" (as reported by (Grad 2002). This definition states clearly that human health is not merely a property of medical doctors. Mental and social well-being refer to the interactions between humans and their biotic (e.g. other individuals) and abiotic environment (e.g. the city they live in, the air they breath) as well as their perceptions and mental states in relation to these interactions. A broad range of disciplines can therefore pretend contributing to human health. Identically the Office Internationale des Epizooties (OIE) not only deals with animal diseases and their impact on animal productions but is also concerned by animal welfare. Given the impact of animal diseases on animal productions and on food security and the impact of zoonoses on human health, more integration is needed between the fields associated with human and animal health. A subset of animal health is wildlife health. Wildlife health can be important for wildlife itself, notably for endangered populations or species and for human and domestic animal health when they are interactions and in the case of multi-host pathogens (Daszak, Cunningham & Hyatt 2000). The link between human and animal health has been observed for a long time (Schwabe 1984) and has recently received much attention through the development of what I will call global approaches to health (GAH) including initiatives/concepts such as One health or Ecohealth (Zinsstag et al. 2011). If these concepts or initiatives have been initially quite well defined, each stakeholder seems to have designed its own definition and often these concepts are perceived loosely, "however, all of us believe it is important (...)" as (Chien 2012) reports when referring to the One Health concept.

Practically, the interdependency between human, domestic animal and wildlife health, especially in SES with extensive wildlife/livestock/human interfaces offers the possibility to capture the interest of all stakeholders when dealing with health. We observed this when implementing animal health protocols at the wildlife/livestock interface under the RP-PCP approach. One can extend the concept of (human) health as a public good (Chen, Evans & Cash 1999) to human and animal health as a shared public good in SES. Often in the literature or in international organisation reports, ecosystem health is added to human and animal health as a third component. I don't think these concepts can easily be put next to each other. They refer to complete different realms. Within the development of GAH, I believe that there is also some fundamental work to be done on the relationship between human and animal health on one side and ecosystem health on the other. "Ecosystem health" is misused in the One Health & EcoHealth literature and often simply displayed in relation to Human and Animal Health as the third object of the Human/Animal/Ecosystem Health without any theoretical background proposed. It seems that using the health of an organism as the metaphor to refer to the state of an ecosystem is the only reason why this term is used. If this concept is quite well developed in the ecological literature (e.g. (Costanza 2012), it is not yet appropriated in the One Health literature and there is a need for a thematic bridge to be developed. An interesting question deriving from the later is to which extent wildlife well-being (part of wildlife health) is linked to the state of a protected area or of a Human/Nature interface and hence linked to ecosystem health? The health of wildlife could in principle be an indicator of ecosystem health along with others such as plant and animal biodiversity for example. They are interesting avenues that need to be explored belonging to very different scientific fields. For example, Māori from New Zealand manage their

water systems from a cultural and societal point of view and a cultural health index has been developed in order to link this traditional management to state management of water source (Tipa & Teirney 2006). This work clearly relates the health of an ecosystem (here centred around the water source) and the mental state and well-being of its users, the Māori. It also indicates how ecosystem health is linked to an anthropological view of ecosystems. I would like in the coming years to explore more what means "ecosystem health" and how we can soundly integrate it to GAH.

C. Transmission Ecology in multi-host systems

1. Infectious disease trends and the need for Transmission Ecology

Recent trends in human Emerging Infectious Diseases (EIDs) indicate that 60.3% of outbreaks concern zoonoses (i.e. diseases transmissible from animal to humans) and among these outbreaks, 71.8% originated in wildlife (Taylor, Latham & Woolhouse 2001; Jones et al. 2008). In domestic stock, 77.3% of pathogens are multi-hosts (Cleaveland, Laurenson & Taylor 2001). Infectious diseases therefore represent a threat for animal and public health as well as for biodiversity conservation when pathogens spillover from domestic stock and human to wildlife. In addition, the majority if not all of host-pathogen systems have to be considered within a broader multi-pathogen environment (Woolhouse 2008), where interactions between hosts, pathogens and other organisms are still hypothesised and little studied (Telfer & Bown 2012). One can therefore expect that the recent and increasing juxtaposition of natural and anthropological systems leading to multiple potential sympatric host populations and their pathogens can predispose to disease emergence. The concept of hotspots of EIDs relates to high-risk areas for pathogen spread and emergence and review studies have identified tropical and sub-tropical regions as potential hotspots for EID (Jones et al. 2008). There, agro-ecosystems where wildlife, livestock and humans strongly interact represent such hotspots for disease emergence and spillover between host species. As human activities increase, the pressure on natural ecosystems through land encroachment, unsustainable use of natural resources and fragmentation of habitats tend to expand (Wittemyer et al. 2008). This trend is exacerbated by human population growth and the need to access more land to feed all (Baudron & Giller 2014; Cumming et al. 2014).

Resulting human/wildlife conflicts can take different forms (e.g. (Hoare 1999; Guerbois, Chapanda & Fritz 2012) that impact human livelihoods and biodiversity conservation. Amongst human/wildlife conflicts, and sometimes not considered as such, the spread of pathogens, other microorganisms and genetic material can represent a burden on wildlife, livestock and human population health (Daszak, Cunningham & Hyatt 2000; Woolhouse 2008; Caron et al. 2013). Diseases significantly impact livestock productions, which are a key livelihood option in semi-arid areas, and they also threaten endangered wildlife species (Kock 2005). In addition, such ecosystems are often characterised by remoteness and poor local development where disease surveillance, prevention and control are weak, creating a local, regional and international risk of disease spread and emergence. In these hotspots, the processes of transmission of pathogens between multiple hosts and the main drivers of emergence have not yet been identified (Childs 2007; Caron, Morand & de Garine-Wichatitsky 2012). This gap in knowledge concerns also endemic and neglected diseases that impose strong constraints on livestock production systems and local livelihoods (e.g. (Hotez & Kamath 2009; Noden & van der Colf 2013; Caron, de Garine-Wichatitsky & Roger 2014). These SES stand therefore as a frontline for global disease emergence surveillance and control but also as the centre of important health issues linked to development and conservation contexts.

For example, in southern African transfrontier conservation areas (TFCAs), people, livestock and wildlife share space and resources in semi-arid landscapes. These TFCA initiatives are expected to increase the land devoted to wildlife activities, a viable land-use option for these arid ecosystems, and will facilitate the movement and mingling of wildlife populations living in protected areas separated by national borders. One consequence of the coexistence of wild and domestic herbivores is the risk of pathogen transmission. Diseases shared by wildlife and domestic animals are an important cause of concern for farmers, veterinary services and conservationists. Human populations living on the periphery of protected areas in southern Africa often rely heavily on livestock production to ensure their livelihoods. In semi-arid and arid areas, where crop failure is common due to erratic rainfall, livestock production assumes an even more important role. However, diseases maintained or transmitted by wildlife can cause mortality and morbidity of livestock, decreasing livestock production (e.g. foot and mouth disease, theileriosis, cowdriosis). Conversely, wildlife species can be affected by diseases infecting domestic animals, which often are imported and therefore are alien species within the ecosystem (e.g. bovine tuberculosis)(Bengis, Kock & Fisher 2002; de Garine-Wichatitksy et al. 2013a). In the case of zoonoses, the health of rural communities with difficult access to health services can suffer from the spillover of pathogens from animals (e.g. bovine tuberculosis, brucellosis, rift valley fever). When wildlife, domestic and human populations interact, opportunities therefore exist for pathogens to emerge in either direction. These risks threaten local livelihoods relying on animal production, public health in the case of zoonoses, national economies in the context of transboundary animal diseases, and the success of integrated conservation and development initiatives (i.e. threat on biodiversity conservation). The level of interaction between sympatric wild domestic and human hosts defines opportunities of pathogen transmission between host populations.

The standard and historical approach to tackle infectious diseases in human and animals is to explore the relationship between a single host and a single pathogen, sometimes including some intermediate host and/or some vector species (Morgan et al. 2004; Caron, Morand & de Garine-Wichatitsky 2012; Daszak et al. 2012). Current medical and veterinary surveillance systems fail therefore to grasp the complexity of EID and other endemic diseases in multi-host multi-pathogen systems. By definition, in the context of EIDs, the new pathogen has not yet emerged or has recently emerged and its ecology (epidemiology) in multi-host systems is unknown. Current surveillance approaches rely on prior detection of the pathogen before action and thus always lag one step behind the epidemiological event (Childs & Gordon 2009; Flanagan et al. 2011). In addition, the global fight against EIDs is more and more focusing on identifying potential emerging pathogens from high-risk maintenance hosts (e.g., bats and rodents)(Calisher et al. 2006; Luis et al. 2013). If such an approach is important and efficient at adding new names to the infectious agents' list, this pathogen hunting does not provide information on which of these micro-organisms might present a significant threat to host populations of interest such as human, domestic or wildlife species. In the same line of thoughts, attempts to identify hotspots of disease emergence at regional scale do not provide a framework to address infectious disease transmission in multi-host contexts within SES, meaning locally. Therefore, if socio-ecological contexts of pathogen emergence and spread can be identified (e.g. tropical socio-

ecosystems with increasing wildlife/livestock/human interfaces), an approach to prevent the diffusion of known and unknown pathogens in multi-host systems is yet to be developed (Caron, Morand & de Garine-Wichatitsky 2012). We argue that such an approach should focus on identifying the functional processes that lead to pathogen transmission between host populations in order to prevent the spillover event between hosts instead of lagging one step behind the epidemiological event as most current surveillance system do. The on-going Ebola outbreak in West Africa stands as a perfect example: the prevention of the spillover from the animal source, be it the probable maintenance hosts, specific bat species, or other hosts such as hunted antelopes, feral dogs or pigs would have saved most of the human and financial costs of this epidemic. The same apply to SARS, H5N1 HPAI but at the same time to more neglected tropical diseases such as brucellosis, tuberculosis and many others.

These interfaces represent complex multi-host and multi-pathogen systems that have been so far little studied (Viana et al. 2014). As already said, the classic veterinary approach has not been designed to apprehend multi-host systems and it needs to be strengthen by ecological sciences, notably community ecology which by definition deals with multiple species in interaction. A more functional approach could be adopted to reduce this complexity by focusing for example on the detection of transmission processes per se. As pathogens can only be transmitted through a limited number of transmission modes (e.g. direct, environmental or vector-borne transmission), an approach that could identify the transmission pathways of micro-organisms between host populations and the environment would help guiding multi-pathogen surveillance and control at wildlife/livestock/human interfaces. For example, the identification of behaviours triggering direct contact between hosts or of spatial attractors such as water point or food resources that increase the utilization of a place by multiple hosts could help to provide mitigation options to reduce host contacts and therefore microorganisms transmission.

I have been developing this approach notably with Michel de Garine-Wichatitky and Serge Morand but also with many other researchers from within our research unit and collaborators from other institutions at least for the past 7 years. I call this thematic field "transmission ecology" as a proxy for "infectious transmission ecology". Investigating transmission pathways between hosts can be summarized as identifying what type of ecological contact or interaction between hosts can lead to infectious contacts. In addition, one can investigate which host and pathogen traits influence the success or failure of inter-host contact to translate into infectious contacts. The advantage of working on transmission pathways between hosts are numerous: 1) by identifying what is currently transmitted between hosts, one can to some extent infer what could be transmitted in the future (e.g. EIDs) given that all pathogens must be transmitted through a limited number of transmission modes (e.g. direct, environmental, food-borne)(Caron, Morand & de Garine-Wichatitsky 2012; de Garine-Wichatitsky, Caron & Morand Submitted); 2) by investigating contacts between hosts that can lead to transmission, one can prioritize species to survey within complex multi-host systems with dozens of species interacting; 3) Prioritizing species within a SES that can threaten the target population (i.e. the population that one wants to protect; human, domestic or wild population) can guide the limited amount of resources that one has to implement surveillance and control of animal and/or human diseases (Caron et al. 2014).

2. Epidemiological function concept

Transmission ecology requires instilling more ecological concepts into epidemiological thinking. Functional ecology, looking at the role or function that an organism plays in an ecosystem unfolds a useful framework (Begon, Townsend & Harper 2006). Community ecology defined as the study of the interactions between species in communities on many spatial and temporal scales, including the distribution, structure, abundance, demography, and interactions between coexisting populations is not only focusing on one organism and its relations with its environments but to the sum of organisms living together in an ecosystem (Begon, Townsend & Harper 2006). Community ecology can therefore help to tackle issues related to pathogen transmission in multi-host systems.

Ecological functional approaches classify organisms according to what they do, and/or what they eat. They offer an alternative perspective to taxonomic classifications for identifying trends within and making sense of ecological complexity. Applications of functional group concepts, which date back to fundamental ideas about biomass distributions across different trophic levels (Elton 1927), have been crucial in advancing ecological understanding. More recently, ecological functional analyses have achieved prominence as a way of linking taxonomic survey data and the provision of ecosystem services (Cumming & Child 2009). Functional analyses thus remain an important research area in ecology (Caron et al. 2015a).

In epidemiology, functional concepts have clear potential utility but are still in a relatively early stage of development. Classical epidemiology relied heavily on single-species studies, particularly those of people (e.g., analyses of measles and smallpox in human populations (Anderson & May 1991). In contemporary epidemiological studies, in the last fifteen years, under the influence of ecology, the scope of epidemiology is being broadened to include plant and animal communities in which multiple different species can contribute to the maintenance and spread of pathogens in host populations (Plowright et al. 2008). In multi-host systems, the role played by each host population in pathogen dynamics is determined by the species' competence for the pathogen (i.e., its receptivity to infection and its capacity to replicate and transmit the pathogen (Komar et al. 2003), its exposure to the pathogen determined by the host ecology/behaviour and its interactions with other host populations (including vectors for vector-borne infections) leading to infectious contacts, and finally, the composition of the host community that will determine the range of inter-host interactions (LoGiudice et al. 2003).

One of the central questions in transmission ecology is that of how the community composition of potential host species relates to the dynamics of pathogen transmission within the host community, as opposed to within a population of a single species. The complexity of this problem can be simplified by assigning epidemiological functions to relevant traits that define an organism's role in the epidemiology of a given pathogen. For example, animals that undertake long movements (a trait) may contribute to the epidemiological function (pathogen disperser) of spreading pathogens over large distances (a role). Grouping organisms by epidemiological functions facilitates the development of eco-epidemiological models for a given pathogen in relation to an entire animal community (Caron,

Morand & de Garine-Wichatitsky 2012). This approach could potentially play an important role in guiding research, as well as in the surveillance and control of animal and zoonotic diseases (Caron et al. 2012b).

First, if numerous hosts exist, they can only play a limited number of roles in the epidemiology of a given pathogen (e.g. maintenance, intermediate, bridge or dead-end host). Thus, a way to reduce hosts' diversity is to focus on their epidemiological functional roles (Caron et al. 2012b; Caron et al. 2014; Caron et al. 2015a; de Garine-Wichatitsky, Caron & Morand Submitted). Secondly, despite the large (and largely unknown) pathogen diversity, pathogens can only be transmitted through a limited number of transmission modes (e.g. direct, environmental or vector-borne transmission). Hence, in principle, transmission modes could be used as a "functional bottleneck" in the epidemiology of pathogens. Epidemiological functional roles and modes of transmission may therefore provide a framework to reduce the complexity of host-pathogen systems in EID hotspots.

What is meant by the "functional bottleneck" role played by transmission modes is that the entire pathogen diversity will have to use one of the few transmission modes available. If these transmission modes' pathways can be identified and/or quantified between host populations in a given ecosystem then a new type of surveillance can be implemented targeting transmission pathways between host populations that could be relevant for an array of pathogens sharing the same transmission modes. For example, if the water-borne transmission pathway is identified as the driver of multiple infections in domestic animals or humans in a given ecosystem, changes in human behaviour and herding practices can be implemented to stop this transmission chain (e.g. by using different surface water sources) leading to a positive impact on multiple infections (Caron et al. 2015a).

Although some progress has been made in the characterization of epidemiological functional groups, (e.g., clear definition of the maintenance function (Haydon et al. 2002; Viana et al. 2014), other epidemiological functions remain incompletely defined, especially those relating to the transmission of pathogens between groups of hosts. I (we of course) have mainly worked on two epidemiological functions in the recent years: the maintenance and the transmission function. In order to define epidemiological functions, I need to use some of the roles that hosts can play (e.g. target, maintenance) before having the chance to unfold their full definition and context (in the "C.3" section). The reader can jump to those parts if more details are needed.

Maintenance function

We use "host" to refer to a host population, a host species, or a host community. The smallest epidemiological unit to which we will refer is a host population, acknowledging the fact that individual variability can also impact substantially pathogen transmission (e.g. "superspreader", (Paull et al. 2012). As defined by Haydon et al. (2002) and more recently revised by Viana et al. (2014), a conceptual framework for the role of hosts in epidemiology requires the definition of the target host: "the population of concern to the observer" in the area under study (see also C.3).

The maintenance function represents the capacity to maintain the pathogen within the ecosystem. A maintenance host is a host population (single population) "in which the pathogen persists even in the complete absence of transmission from

other hosts" (Nugent 2011)(see C.3). A maintenance function has been proven for several populations at wildlife/livestock interfaces: domestic dogs for Canine distemper and Rabies viruses in the Serengeti ecosystems (Prager et al. 2013; Viana et al. 2015); European badgers (*Meles meles*) for bovine tuberculosis (bTB) in the United Kingdom (Scantlebury et al. 2004); brush-tailed possums (*Trichosurus vulpecula*) for bovine tuberculosis in New Zeland (Nugent 2011); African buffalos (*Syncerus caffer caffer*) for bovine tuberculosis and foot and mouth disease (FMD) viruses in southern Africa (Bengis, Kock & Fisher 2002); red foxes (*Vulpes vulpes*) and rabies in Europe (Freuling et al. 2012); Anseriformes (ducks and geese) for low pathogenic avian influenza, worldwide (Webster et al. 1992).

More than one host population of different species can constitute the maintenance function. Two non-maintenance populations can when they are sympatric and in interaction create a "maintenance community" where the pathogen can be maintained (Haydon et al. 2002). In multi-host systems, a maintenance community needs to be suspected when closely related species coexist as in avian communities (Caron et al. 2012b).

Transmission function

Although the maintenance-target host relationship and its link with the maintenance function have been properly defined, the function of pathogen transmission to the target host needs a clearer definition. Interspecific pathogen transmission is of crucial importance for infectious disease management. Disease control can target the maintenance host to stop pathogen maintenance and circulation in the ecosystem (i.e. targeting the maintenance function); however, as this option is often unfeasible (for practical or ethical reasons, notably concerning wildlife populations), one could also try to break the transmission pathway that brings the pathogen to the target host (Caron et al. 2015a). We therefore define *the transmission function* as the capacity to transmit the pathogen to the target host. This function must be separated from the maintenance function, as the maintenance host does not always have infectious contact with the target host. When it has direct contact with the target host, then the maintenance host is implicated in the maintenance and transmission functions. When it does not, a bridge host can connect (i.e., have infectious contact with) both maintenance and target hosts, "bridging" the gap between them.

Defining epidemiological functions such as the maintenance and transmission function can help grouping hosts into epidemiological functional groups that share a role in epidemiological processes, framing a conceptual epidemiological model that can be tested and revisited if needed (Caron et al. 2014; Caron et al. 2015a; de Garine-Wichatitsky, Caron & Morand Submitted).

3. Epidemiological functional groups

Applying the functional approach presented previously, we develop the concept of Epidemiological Functional Groups (EFGs), to which species may be allocated according to the functional role they play in pathogen transmission. Although numerous potential host species/populations may coexist in transmission hotspots, they can only play a limited number of roles in the epidemiology of a given pathogen, or of a group of pathogens characterized by similar modes of

transmission and host range. Table 1 summarizes the roles that host populations may play in the epidemiological processes leading to the emergence of a pathogen from maintenance to a defined target population.

Target EFG

As defined in Haydon et al. (2002), an epidemiological functional framework cannot be implemented without having a surveillance or control objective. This objective is motivated by a host population "that one wants to protect" or target population or host. For public health professionals, the target host is represented by a human population at risk of being infected by a zoonosis when in contact with source animal populations. For veterinarians, domestic animal populations are the target hosts. For wildlife health professionals, a vulnerable wildlife population can be threatened by a pathogen spreading in the wildlife community or in contact with domestic or human populations hosting hazardous pathogens. In endangered wildlife populations, diseases has been shown to be agents of extinction (Smith, Sax & Lafferty 2006).

The necessity to define a target population translates the applied approach that we want to develop. *In fine*, the surveillance and control activities that one want to implement can only be done in relation to this target population. In addition, different target populations can have different maintenance host populations. For example, cattle can be a maintenance host for *Mycobacterium bovis*, the agent of bTB for the human target host (Biet et al. 2005) and brush-tailed possum can be a maintenance host of *M. bovis* for cattle (Nugent 2011). Of course, the fact that cattle can maintain bTB will have to be taken into account even if cattle the target host but the way surveillance and control will be implemented could differ if the activities aim at controlling or preventing bTB to spillover in cattle or in human.

Table 4: Role of hosts in transmission ecology

Role of hosts	Definition or related definition for the case of bridge host	Maintenance	Transmission	Examples
Target host	- The population of concern to the observer (Haydon *et al.* 2002)	X		- Human populations (for zoonoses) - Domestic populations - Threatened wildlife species
Maintenance host population	- Hosts in which the pathogen persists even in the complete absence of transmission from other hosts (Nugent 2011) - Population larger than the critical community size (i.e. size under which the pathogen cannot be maintained in the community) in which the pathogen persists (Haydon *et al.* 2002)	X	(X)	Brush-tailed possums for bovine tuberculosis in New Zealand (Nugent 2011)
Maintenance host community/ Maintenance host complex	- One or more epidemiologically connected populations or environments in which the pathogen can be permanently maintained (Haydon *et al.* 2002) - Any host complex in which disease persists indefinitely is a reservoir (Nugent 2011) - Host for which cross species transmission and inter-species transmission are high (Fenton & Pedersen 2005)	X	(X)	Dabbling ducks for avian influenza viruses in Africa (Gaidet *et al.* 2012a)
Bridge host	- Non-maintenance host population able to transmit a pathogen from a maintenance host/complex to the target population, otherwise not or loosely connected to the maintenance complex**.** Previous related definitions: - *Source population*: any population that transmits infection directly to the target population (Haydon *et al.* 2002) - *Liaison host*: incidental hosts that transmit pathogens from a reservoir to another incidental host (Ashford 1997; Ashford 2003) - *Spatial vector*: host that transport the pathogen to target populations in new locations (Nugent 2011) - *Temporal vector*: host that can transmit the pathogen to target species across temporal scale (Nugent 2011)		X	- Little studied so far - Peri-domestic birds such as swallow sp., sparrow sp., etc. (Caron *et al.* 2014)

Crosses in brackets indicate that maintenance host can participate in the transmission function although this is not a necessary condition. (Caron *et al.* 2015a)

Reservoir

I would state (and this is a personal opinion compared to the shared framework presented here) that the concept of "reservoir" in epidemiology and disease ecology is often misused and a reference should at the minimum be associated with its use. I would tend not to use it as it is connoted with too many meanings. There are many definitions of reservoir existing. Table 5 presents a few of these definitions.

Table 5: Definitions of reservoir

1. The habitat in which an infectious agent normally lives, grows and multiplies; reservoirs include human reservoirs, animals reservoirs, and environmental reservoirs	**CDC website; http://www.cdc.gov/ophss/csels/dsepd/ss1978/lesson1/section10.html**
2. Any person, animal, arthropod, plant, soil or substance (or combination of these) in which an infectious agent normally lives and multiplies, on which it depends primarily for survival, and where it reproduces itself in such manner that it can be transmitted to a susceptible host	ULCA School of Public Health http://www.ph.ucla.edu/epi/bioter/anthapha_def_a.html
3. The long-term host of a pathogen of an infectious disease	(Janeway et al. 2001)
4. One or more epidemiologically connected populations or environments in which the pathogen can be permanently maintained and from which infection is transmitted to the defined target population	(Haydon *et al.* 2002)
5. An ecologic system in which an infectious agent survives indefinitely	(Ashford 2003)
6. If R0>1 for a pathogen in a host population	(Nugent 2011)
7. Wild species capable of carrying infections transmissible to humans	(Han *et al.* 2015)
8. A population which is chronically infested with the causative agent of a disease and can infect other populations	Oxford English Dictionary - medical term
9. A place or container used for the collection and storage of water, other fluids, or even solid material	Oxford English Dictionary - general definition

(Caron, unpublished data)

First, many of these definitions use a vague terminology not adapted to disease ecology in multi-host systems as we see it today. For example in definition (1) & (2), the term "normally" is difficult to interpret. How is "normally" defined in epidemiological terms? And in the context of global change and recently created wildlife/domestic interfaces where norms are changed? Definition (3) refers to "long term" which does not mean anything in terms of maintenance. The Medical version of the Oxford English Dictionary (definition 8) only deals with single population and therefore does not account for maintenance communities.

Second, in multi-host systems, the reservoir concept is still debated and used in contradictory ways, as discussed by several authors (Haydon et al. 2002; Ashford 2003; Nugent 2011). Haydon et al. (2002) extended the definition of reservoir by adding "source populations" (Table 4) that may not be involved in the maintenance of the pathogen but rather in the transmission of the pathogen to the target population (definition 4). Ashford (2003) (definition 5) defined a "liaison host" as linking the reservoir to another host population, with no explicit reference to target populations. We agree with Ashford (2003) that source population should not be included in the definition of the reservoir, as this later term is strongly linked to the concept of maintenance and because control of infection in the reservoir would be different if targeted at the maintenance or source populations. For example, aiming at controlling the infection in a maintenance *vs*. a source population might have different outcomes, as the maintenance host could still re-infect the source

population in the latter case. Nugent (2011) (definition 6) provides a mathematical definition of reservoir using the concept of R0. However conditions on R0 (e.g. initial host population is fully susceptible) do not account for long-term maintenance of the pathogen within the host population.

Third, the concept of the reservoir as revisited by Haydon et al. (2002) and more recently by Viana et al. (2014) does not refer clearly to a single epidemiological function as we defined them, because it includes maintenance host(s) involved in the maintenance function and potentially in the transmission function as well as non-maintenance population(s) only involved in the transmission function. Allocating reservoir hosts to specific functional groups that surveillance and/or control can target is therefore difficult and provides an additional reason to focus solely on the maintenance-target hosts.

Fourth, if the metaphor used for "reservoir" is appealing for the concept of "maintenance" for a pathogen, it is misguiding as Ashford (Ashford 2003) mentions, because it also refers to the static notion of a "tank" or "storage" (definition 9) which does not illustrate the dynamics of transmission necessary for the maintenance function to operate, especially in multi-host systems.

As a result, "reservoir" is misused in the literature. Given most of the definitions existing (see Table 5), only a few authors refer to the definition they use. Others give their own definition (see definition 7: a sero-prevalence is enough to statute on the capacity of reservoir in (Han et al. 2015). And some refer to a particular definition but fail to follow it. For example, Suzán et al. (2015) have presented a new framework to understand patterns in space and time of meta-communities of hosts and parasites. In their first figure they display in red "reservoir species" and in orange "alternative hosts", together "maintaining higher infection of prevalence". Clearly, their concept of "reservoir" differs from Haydon et al. (2002), who argued that any host involved in the maintenance of the pathogen should be part of the reservoir. The difference in definitions is identical with Plowright et al. (2015): they present domestic horses as potential source populations (defined in the article as "recipient" and "intermediate hosts") of Hendra viruses for human populations without considering them as part of the reservoir (presented as the bat community). These examples are not exceptions: most of articles using the term "reservoir" use it improperly giving their reference definition if any.

Finally, confusion is sometimes made due to lack of semantic between "reservoir" and other epidemiological concepts. For example, with "superspreader" (the capacity of an individual, a population or a community to initiate relatively many secondary infections (Paull et al. 2012); as well as concept such as "hyperreservoir" defined sometimes as the capacity for a species (not a population) to host more than 2 species of pathogens (with no mention of maintenance)(Han et al. 2015). In this last example, there is a clear confusion between pathogen-level and pathogen community-level properties.

The extensive use of the "reservoir" concept under multiple definitions and the lack of consensus around the liaison host and source population concepts (revealed by the scarcity of use of these two last terms in the literature) requires a refined conceptual framework and definitions. Agreeing with others (Ashford 2003; Nugent 2011), we thus prefer to use only maintenance host or community, a term that refers better to the dynamic aspect of the functional role than the static notion of a reservoir (LoGiudice et al. 2003; Johnson et al. 2013). The only case for which

reservoir could be used is in the case of maintenance of the pathogen in the environment, a so called "environmental reservoir in definition 1 in Table 5: this type of maintenance implies a survival of the pathogen without any transmission event and refers quite well to Oxford English Dictionary definition 9.

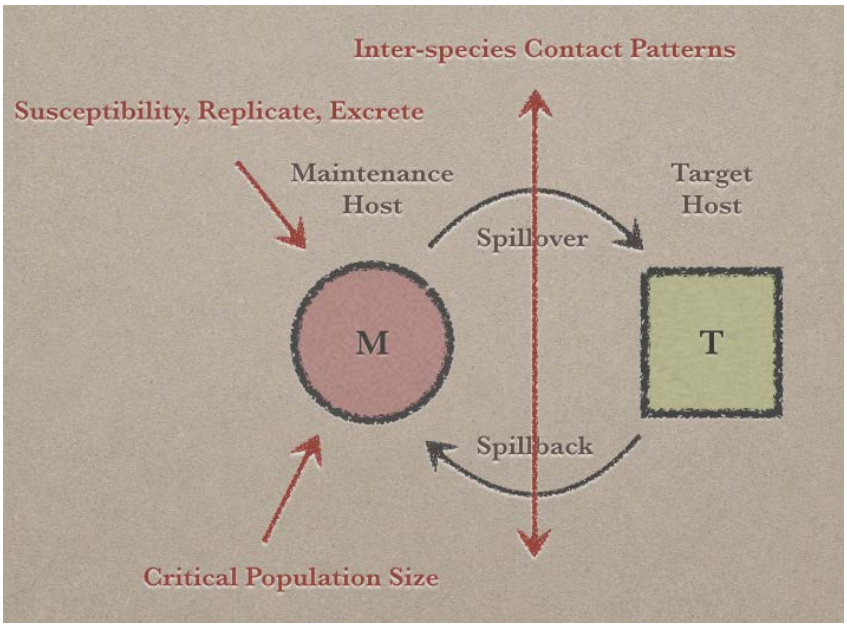
Maintenance EFG

A maintenance host is a host population (single population) or community/host complex (several sympatric host populations) "in which the pathogen persists even in the complete absence of transmission from other hosts" (Nugent 2011)(Table 4). This definition of EFG is linked with the maintenance function.

The maintenance function depends on a range of factors presented in Figure 4:

- Host density and abundance will determine if the population reaches the critical community size under which the pathogen cannot be maintained. The critical community size definition states that there is no "maintenance species" *per se* and that there will always be a population size under which the host will not be able to maintain the pathogen (Lloyd-Smith *et al.* 2005). The notion of maintenance can therefore only be referred to a particular host population in a given ecosystem or set of ecosystems. For example, European badgers are maintenance hosts for bTB in the UK but their role in the rest of Europe is still unknown and not assumed to be a maintenance host without more investigation.
- Host susceptibility to the pathogen and its capacity to replicate and excrete it what I called "host competence" (Caron *et al.* 2014).
- Intra- and inter-host infectious contacts (i.e., a contact leading to infection amongst other intra-host factors; (Fenton & Pedersen 2005).

Figure 4: Maintenance and target hosts



Red arrows indicate factors influencing the maintenance function

The capacity to maintain a pathogen is therefore a fine balance between the impact of the pathogen on its host, the host population size and the connectivity of

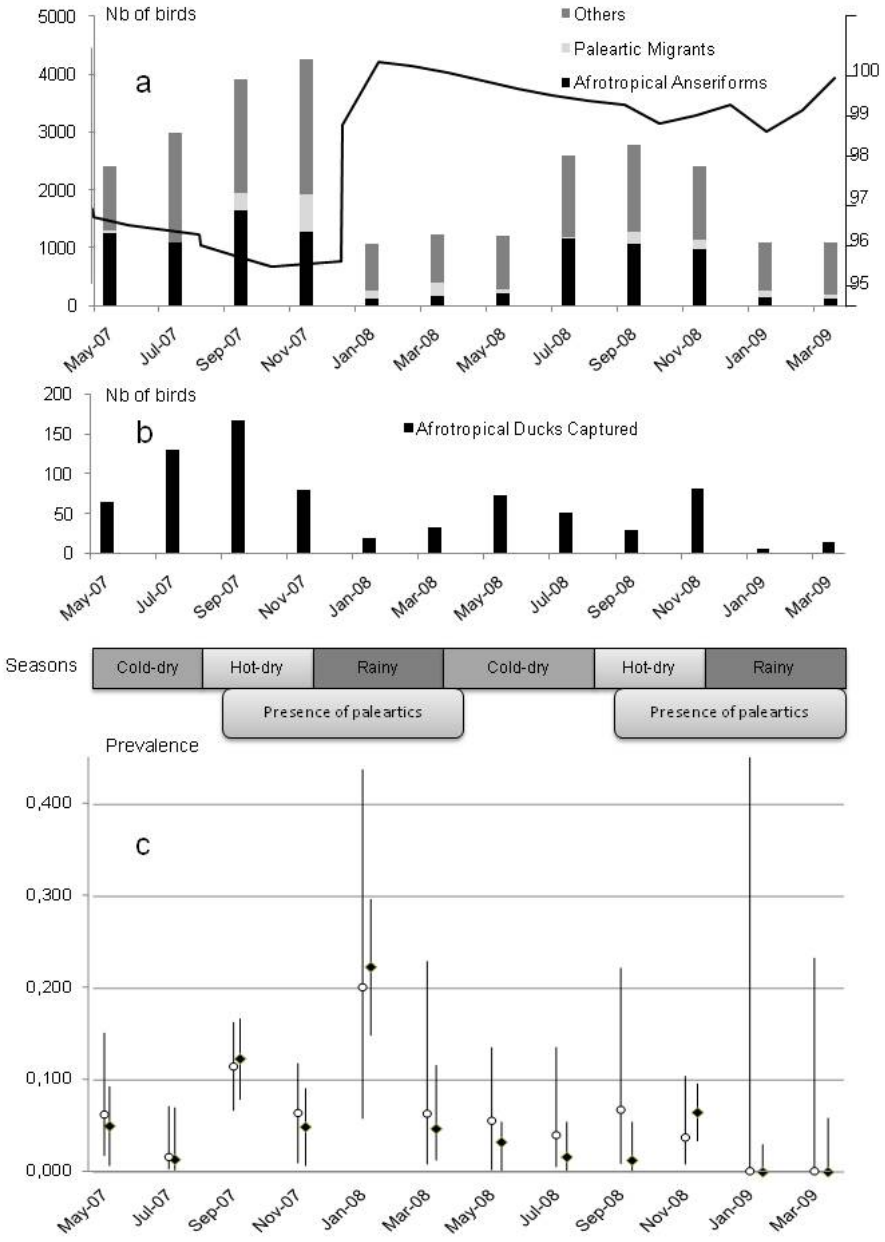
individuals (and populations when referring to host communities). In theory, a pathogen killing its host, even quickly, can still be maintained within this host population or community if the host population is large enough and the network of contacts between hosts is frequent and dense enough to prevent the pathogen dying with its individual host.

The use of "maintenance" instead of "reservoir" is made easy because this term is already widely used in the literature and there is no controversy about its definition. Most people mean "maintenance host" when they talk about "reservoir", when according to Haydon (2002) "reservoir" includes also species not maintaining the pathogen. And finally, the notion of "maintenance" does not refer to the notion of a static container, as does "reservoir" in the definition of the Oxford English Dictionary.

Maintenance vs. persistence

There is a clear distinction to be made also between maintenance and persistence. We defined "persistence" as the capacity of a pathogen to circulate in a host population or community but not being maintained without a regular re-introduction from the outside. The concept is necessary when considering complex systems including multiple hosts.

Figure 5: LPAI prevalence and driving factors in a Zimbabwe ecosystem



Birds counted per session: in black duck species, in light grey Palearctic migrants, and in dark grey other species. The solid line (linked to the right vertical axis) represents the variations of the lake level. b Birds captured per session: in black duck species, in light grey Palearctic migrants, and in dark grey other species. c Global (black dots) and duck (white dots) prevalence per session with confidence interval. An indication of the seasons in this ecosystem is given in blocks; the period when Palearctic migrants are present in the system is also presented (Caron *et al.* 2011).

I had to take into account this concept when investigating the circulation of low pathogenic avian influenza viruses in a wild bird community in a lake ecosystem in Zimbabwe (Caron et al. 2012a). After regular (every two months) testing of wild birds against LPAI for two years, we observed peaks in LPAI prevalence followed by a decrease in prevalence, and peaking again the next year at the same period (Caron et al. 2011). Lake levels variability between the two years of study differed significantly which had an impact on the wild bird community present at the lake, notably during the dry season when we observed a peak in prevalence the previous

year. This second year, the peak in LPAI prevalence was much lower and we did not observed any circulation of LPAI during the following next season. These results suggested a persistence of LPAI in this African ecosystem with the need for a seasonal re-introduction of LPAI, potentially from Palearctic migrants arriving during the European fall in southern Africa. An alternative hypotheses would be that a meta-population system in southern Africa drives the re-introduction of LPAI, notably through nomadic duck populations (Dodman & Diagana 2007) as our results at the southern African scale suggested (Cumming et al. 2011).

We could not confirm this hypothesis notably because of the lack of genetic results. Most of our PCR positive results did not give any information about the LPAI strain and therefore we could not compare the strains isolated in our ecosystem with other strains (e.g. from Europe) and assess their relatedness. This hypothesis is still relevant today and recently "African" LPAI genes have been shown to circulate for extended period of time in South Africa with however the possible circulation as well of "European" genes (Abolnik et al. 2010).

Bridge EFG

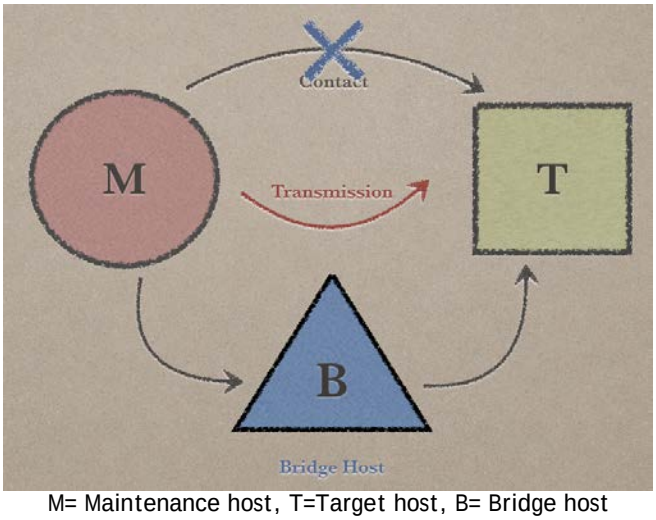
After working on the maintenance function and EFG and because of my work on LPAI in wild birds across Africa and particularly in Zimbabwe, it became necessary to brainstorm around a new EFG. From field observations and given the extensive multi-host system presented by avian communities, the simplification of the disease ecology framework to target populations (i.e. domestic poultry populations, including in southern Africa ostrich farms) and maintenance populations restricted to Anseriformes and Charadriiformes formally recognised as the maintenance hosts for LPAI was not enough. In the SES I came to work in (Mali, Niger, Chad, Zambia, South Africa and Zimbabwe and even Ukraine through the FAO TCP project (Gaidet et al. 2007a; Gaidet et al. 2007b), contacts between the maintenance and the target population where not always obvious. The wide range of LPAI susceptibility in wild birds (more than 100 species according to Olsen et al. (2006) and the opportunity for other avian populations to play a local role in AIV ecology led me to try to investigate this potential epidemiological transmission pathway. I developed this approach in 5 papers, the first one introducing the framework to investigate disease ecology in multi-host systems (Caron et al. 2009), followed by two field study articles (Caron et al. 2010; Caron et al. 2014) and a final review paper unfolding the new bridge EFG and applying it to other multi-host systems and pathogens (Caron et al. 2015a).

When the maintenance host or community has direct contact with the target host, then the maintenance host is implicated in the maintenance and transmission functions. When it does not, a bridge host (Table 4) can connect (i.e., have infectious contact with) both maintenance and target hosts, “bridging” the gap between them (Figure 6).

Bridge host is therefore used, since (i) the group is distinct from the “source population” presented by Haydon et al. (2002), as bridge hosts do not belong to the maintenance host/community, and the “liaison host” presented by Ashford (2003) as a bridge host is always in reference to a maintenance-target population system; and (ii) the word "bridge" is meaningful to the definition proposed (e.g. (Columba Teru et al. 2012). Bridge hosts refer therefore to a group of hosts that perform the same epidemiological function for a pathogen that can be targeted by specific

surveillance and control interventions. In Suzán et al. (2015), information about whether alternative hosts function as bridge hosts would add an important layer of information to their framework and contribute to the understanding of the spatial spread of parasites.

Figure 6: Bridge host in a Maintenance-Target system



M= Maintenance host, T=Target host, B= Bridge host

Our bridge host definition is closely related to the "spatial and temporal vector" concepts presented by Nugent (2011) but unifies them with previous definitions (see above) and broadens them. A bridge host can be defined at the level of a population or a community. Bridge hosts may be frequent in disease ecology, but this term has not been explicitly defined and its usage is not common when referring to the transmission function without any role in maintenance function. For example, it would be incorrect to use the term "bridge species" as the role of a bridge host would refer to a specific host population in interaction with other hosts in a given ecosystem (e.g. contact with maintenance or target populations) and at a specific density (Nugent 2011); the host density and the network of interaction between these hosts in another ecosystem would likely be different and would make it unlikely that a species can play the same epidemiological functional role across its range.

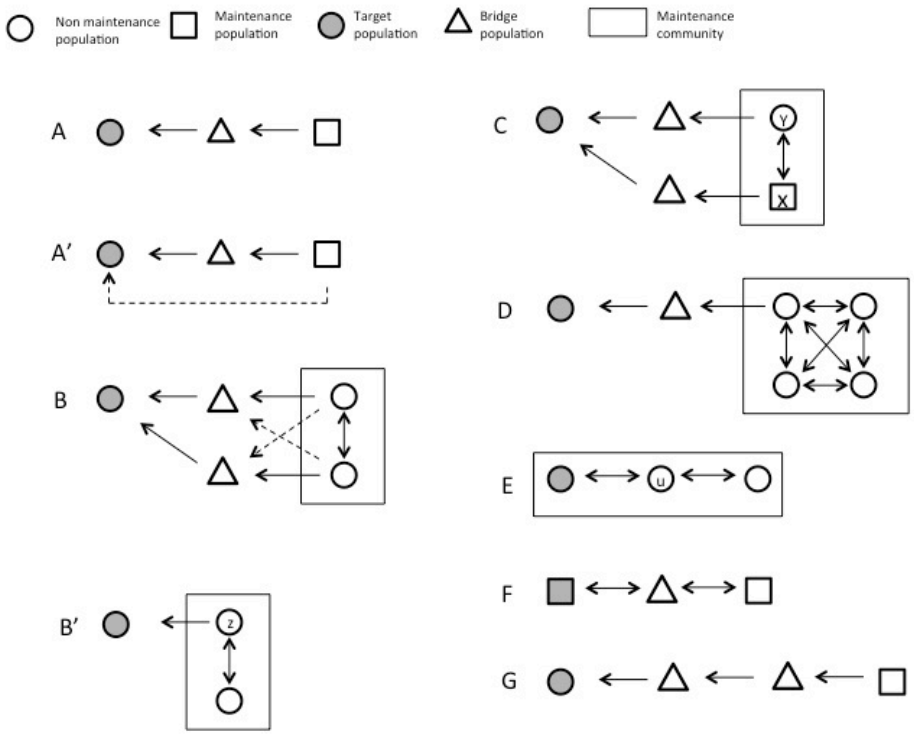
A clearer conceptual framework is thus needed to guide the identification of bridge hosts and the characterisation of their roles in disease ecology. This framework must also be operationalized if it is to guide the design of hypotheses that can be tested through field protocols to characterise the role(s) of hosts in disease ecology.

Using the different target-maintenance systems proposed by Haydon et al. (2002), bridge hosts can be included in target-bridge-maintenance systems in several ways (Figure 7). According to our definition, a bridge host is involved in the transmission function while not involved in the maintenance function. Two main prerequisites must be fulfilled for a host to qualify as a bridge host. The first prerequisite is that the host must be competent for the pathogen (i.e., must be receptive to infection, permit pathogen replication, and be able to excrete it) without being able to maintain it alone, in which case the host would be considered as a maintenance host; or alternatively, the host should be able to mechanically transport the pathogen (Houston & Cooper 1975; Barin et al. 2010).

Its competence will influence the capacity of a bridge host to achieve the transmission function: if the bridge host has a short pathogen excretion period, it will be able to transmit the pathogen to a target population only if the time lag between contact with a maintenance and then a target host is shorter than the excretion period, or if the distance between target and maintenance is shorter than the maximum distance that the bridge host can travel during its excretion phase. Similarly, for mechanical transmission, the survival of the pathogen on/in the host body part (e.g. skin, hair, mouth, feathers) exposed to the external environment will determine for how long the host can play the bridge role.

The second prerequisite is that infectious contacts must occur along the maintenance-bridge-target transmission chain. These will depend on direct and indirect (e.g. environmental transmission) contacts, the mode of transmission of the pathogen, and the site of infection. The basic reproductive number R0 for the bridge host (not considering here mechanical transmission) should be <1 as it cannot maintain the infection but its force of infection, dependent on the number and extent of infectious contacts with the target host, can be high.

Figure 7: Definition of different target-bridge-maintenance systems



This Figure is adapted from Haydon et al. (2002). A represents the simplest maintenance-bridge-target system. In A', the maintenance and target populations are less connected (frequency/intensity of infectious contacts) than between the maintenance-bridge-target populations. In B, mitigation strategies aimed at one bridge host cannot fully control pathogen transmission to the target host because of the alternative bridge host's pathway. If both maintenance populations were in contact with both bridge hosts (i.e. if dashed arrows exist), controlling contacts between the target population and bridge hosts should be simpler than other control options. In B', according to our definition, Z is not considered as a bridge population as it belongs to the maintenance community. In C, stopping contacts between the maintenance population and the target population by acting on one of the two bridge hosts would not be enough to stop transmission, which can still occur through the second bridge host.

D is a special case of B, understanding the complexity of the maintenance community is not necessary to control the pathogen transmission risk to the target population, which can be achieved through the control of arrows connecting the bridge host. In E, none of the host populations cannot sustain the infection by itself and according to our definition, u is not considered as a bridge population as it belongs to the maintenance community. In F, the bridge host connects the target population with another maintenance host creating a system with a maintenance meta-population, which could change the epidemiological dynamics of the system and the probability of success of intervention strategies (e.g. vaccination coverage to achieve control of the infection in the target population). G is a special case where two bridge hosts are necessary to achieve the transmission function. Good knowledge of the ecological interactions in the ecosystem will be necessary to identify such complex interactions between bridge hosts (Caron *et al.* 2015a).

A bridge host that compensates for a lack of infectious contacts between maintenance and target hosts can operate across different dimensions: spatial, temporal, and behavioural. The spatial dimension arises when the bridge host creates a spatial link between the separate areas in which the maintenance and the target host populations occur. This dimension typically refers to the situations developed below for wild birds and avian influenza. It has been defined as a "spatial vector" by Nugent (2011) when considering the role of feral pigs (*Sus scrofa*) in the epidemiology of bTB in New Zealand.

The temporal dimension arises when the pathogen can persist (but not be maintained indefinitely) in the bridge host for a period of time longer than in the maintenance host or during a distinct season; this has been well described by Nugent (2011) as a "temporal vector", for example when red deer (*Cervus elaphus*) transmit *Mycobacterium bovis* to possum populations that are controlled to levels that are well under the critical community size for bTB maintenance.

The behavioural dimension exists when the absence of contact between sympatric maintenance and target hosts is compensated for by another host that has infectious contacts with both. Situations may occur in which the microhabitat preferences and behaviours of maintenance and target hosts mean that they do not come into direct contact despite using the same locations on a daily basis. Bats, for example, are believed to be the maintenance host for Ebola, and can be sympatric with people; but Ebola transmission from bats to humans is enhanced by the great apes (whose susceptibility to Ebola seems to indicate that they are not maintenance hosts) which feed with bats and are fed upon by humans (Gonzalez, Pourrut & Leroy 2007). It is interesting to note that in all cases, even a R0 close to zero (approximating a dead-end host) could still be important for the transmission function: the capacity to excrete the pathogen for a few hours, associated with some form of dispersal, may be sufficient for a bridge host to come into contact with the target host and infect it. For pathogens like Ebola, the range of hosts that is classically considered to be important in disease ecology may have to be broadened by including hosts that are able to transmit the pathogen over short time- and space-scales. These hosts are commonly considered as playing no role in pathogen ecology and are called dead-end hosts (e.g., most wild avian hosts for avian influenza virus - AIV - apart from Anseriformes and Charadriiformes). Amongst the multitude of those dead-end hosts, the bridge host perspective can identify some that do play a role in disease ecology.

The concepts of transmission function and bridge host contribute to a better understanding of disease ecology in multi-host systems by clarifying the epidemiological processes that are relevant for disease transmission and

maintenance. This perspective fits better with the way that people operationalize complexity theory and makes it easier to develop models of these systems. When maintenance and target hosts are not in direct contact, pathogen transmission relies on successive infectious contacts along the chain of maintenance, bridge and target hosts. Bridge hosts can play a pertinent and legitimate role in disease ecology and could become the targets for surveillance and control for some multi-host systems. For example, in some ecosystems, domestic bird populations are rarely in direct contact with wild waterfowl populations but phylogenetic analyses have indicated that most precursors of HPAIV in gallinaceous poultry have originated from wild waterfowl (Campitelli et al. 2008), suggesting that bridge hosts play a role in AIV transmission at the wild/domestic bird interface. More recently, evidence supporting a role for some passerines (finches, sparrows) in the transmission of the avian-origin human influenza A (H7N9) to human and poultry in China (Jones et al. 2014) suggests a potential role for passerines as bridge hosts between poultry and humans.

The functional approach emphasizes the need to focus on transmission pathways between hosts (and their directionality) instead of relying solely on intrinsic host properties (e.g. density, shedding capacity)(Previtali et al. 2012; Streicker et al. 2013). The presence of a target host defines directionality in the transmission processes and implies a network of inter-connected hosts with different epidemiological roles. Our framework thus provides a better empirical approach to some kinds of epidemiological problem, such as the risk of spread of a specific pathogen towards a target population or the potential for disease emergence in emerging disease hotspots.

The identification of bridge hosts for a given pathogen in a given ecosystem has consequences for disease management, surveillance and control. Once bridge hosts are known, managers can adopt mitigation strategies specifically aimed at reducing contacts between the target and the bridge populations. In the case of AIV, this mitigation can be achieved through strengthening biosecurity measures or decreasing the quantity of attractors on the farm (e.g. water sources or open feedlots)(Caron et al. 2014). The adoption of adequate management measures targeting contacts between maintenance, bridge and target hosts is also more environmentally acceptable than controlling (wild) host populations.

Our conceptual framework helps with framing hypotheses based on current knowledge and using empirical tests to either confirm these hypotheses or call for a revision of our understanding of the epidemiological system (e.g. this host is not a bridge host and therefore has no [or another] role in the local context). However, proving that a bridge host in a complex multi-host system where maintenance communities are composed of numerous interacting populations does not take part in the maintenance function (i.e. that removing the bridge host will not drive the pathogen to extinction, according to Haydon et al. (2002) may necessitate an experimental design that would be difficult to achieve in practice (Viana et al. 2014). In addition, only cases in which maintenance and target populations are not in contact have been considered so far. If they are loosely in contact (case A' in Figure 7), the frequency and efficacy of contacts between different pairs (maintenance-target, maintenance-bridge and bridge-target) would need to be weighted against each other. Decreasing the maintenance-target contacts through management will reveal the relative importance of bridge-target contacts and

could require interventions in order to efficiently stop pathogen transmission (as in the case of control of possums for bTB in New Zealand mentioned earlier). Finally, we have assumed that a bridge host must be competent for the pathogen but in some cases simple mechanical transmission (e.g., a bird carrying the virus on its feathers (Lebarbenchon et al. 2013) may be possible, relaxing the prerequisite on host competence for the bridge host.

The development of complex human/livestock/wildlife interfaces, due to the encroachment of human activities within natural ecosystems, triggers new epidemiological dynamics that may permit a range of wild or domestic bridge hosts to link maintenance communities with new target hosts (Kock 2005). We would expect that domestic species and newly farmed or traded wildlife species would increasingly play bridge host roles in the emergence of new zoonoses. The epidemiology of Ebola, SARS, Lyme disease, and H1N1 AIV, for example, are not yet fully understood but are known to involve multiple hosts. We believe that introducing our definitions and operational framework into research and surveillance could contribute to more efficient use of resources to fill some knowledge gaps.

Our approach builds on that of Haydon et al. (2002) and refines it to take into account potential circumstances under which an extra conceptual development is necessary. Whether this extra development will be necessary in many multi-host systems or will be used only under exceptional circumstances will be answered by studies to come. The examples given here indicate that they could be used for at least a few important diseases. The recent appearance in the epidemiological literature of similar concepts (Burns et al. 2012; Columba Teru et al. 2012; Caron et al. 2014) that are not always placed soundly within a conceptual framework and/or ignore previous definitions suggests also the need for a consolidated review and refinement of these concepts and definitions.

Other EFGs?

A part from the three EFGs presented above, we (Michel de Garine-Wichatitsky, Serge Morand and myself) have been developing other EFGs. By default, hosts that are not connected in the multi-host system but are present in the SES are defined as "not connected". They constitute an EFG (they have "no role") but their presence needs to be acknowledged as changes in the SES (e.g. modification of the wildlife/livestock interface) could change their role and shift them from one EFG to another.

Pathogen transmission from maintenance to target hosts may also be regulated by other host populations at the SES level, by means of various mechanisms (Keesing, Holt & Ostfeld 2006). We could combine these regulatory effects under a regulatory function altering the risk of pathogen spillover to the target host. It would basically operate in two different ways, either by altering the infection risk of maintenance and bridge host (e.g. very competent hosts that amplify the pathogen) or by modifying the population dynamics or the distribution of maintenance or bridge hosts (e.g. predators of maintenance or bridge hosts).

Those hosts "altering the infection risk of maintenance and bridge host" would be susceptible to the pathogen, not participating in the transmission function (hence transmission to the target host as defined) but involved in the pathogen transmission dynamics within the SES. The effect of this intervention into the

pathogen transmission dynamics can have dilution or amplification effect. The amplification effect of this regulatory function has been investigated for some disease systems such as for Lyme disease in the United States (LoGiudice et al. 2003). Tree squirrels (gray squirrels *Sciurus carolinensis* and red squirrels *Tamiasciurus hudsonicus*) have been shown to have a dilution potential on the risk of Lyme spillover to human populations through diverting tick bites from the reservoir populations of white-footed mouse (*Peromyscus leucopus*). Alternatively, a host community dominated by the most competent maintenance host such as the white-footed mouse will amplify the risk of spillover to the target host.

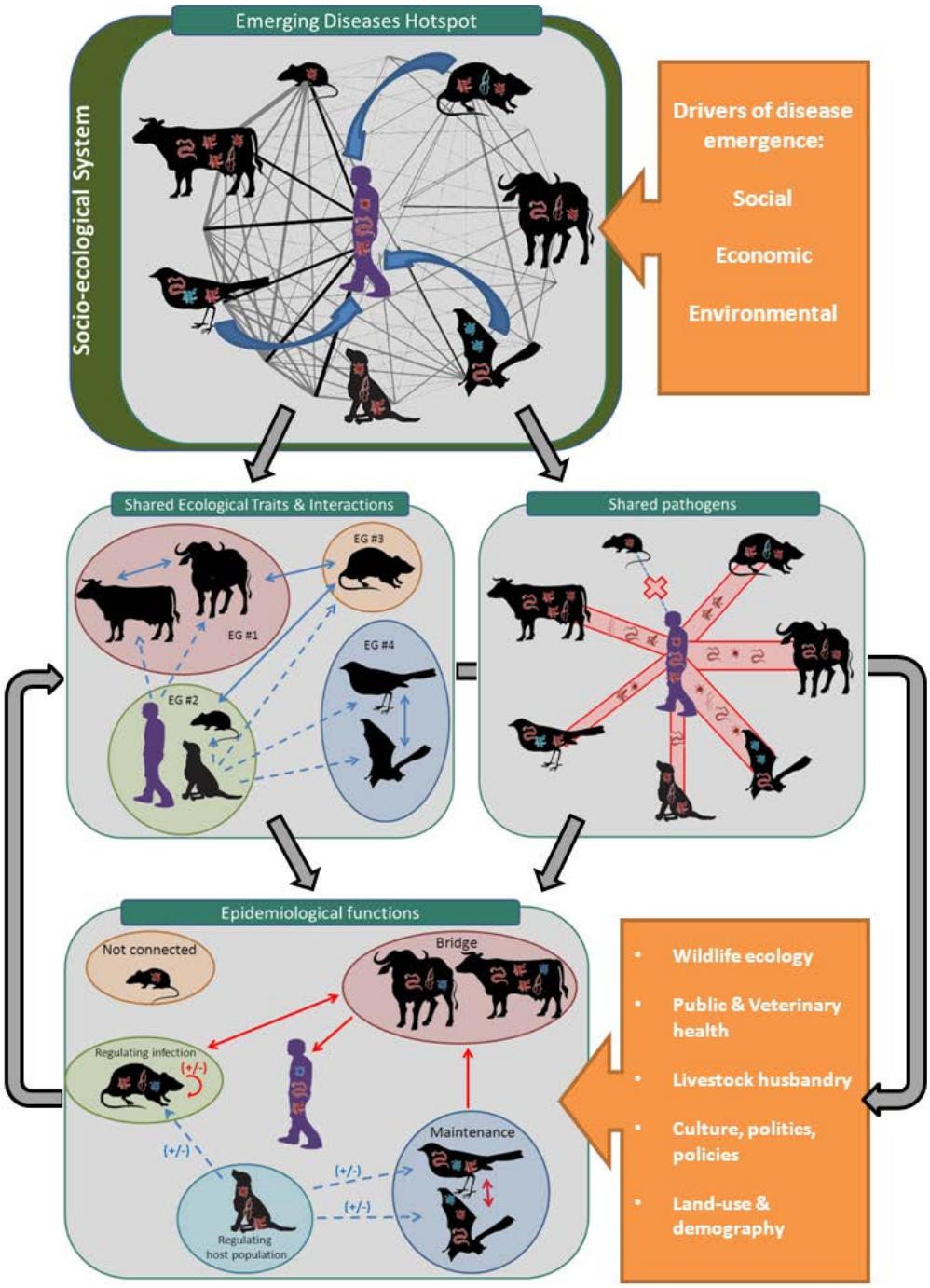
Alternatively, the risk of spillover to the target host can be modulated by ecological interactions between hosts such as predation, competition or parasitism. Those actors in disease ecology have probably been less documented and integrated in epidemiological systems. Predation on some hosts can naturally regulate the host populations and have an impact on the transmission dynamics and ultimately the risk of spillover to the target host. Outcomes of competition processes between a host and another population can also influence the ultimate risk of pathogen spillover. Finally, co-infection states, a common natural situation for host individuals and populations result in direct and indirect (e.g. through the host immune system) interaction between pathogens that can result in changes in the transmission dynamics of the focal pathogen.

The regulatory function is the object of intense debate in the scientific community notably in the field of Biodiversity & Health. The central question is: does biodiversity contribute to an increase or a decrease to the risk of pathogen spillover to target host such as livestock or the human populations (Patz et al. 2004; Pongsiri et al. 2009; Lafferty 2012; Lafferty & Wood 2013; Ostfeld & Keesing 2013; Keesing & Ostfeld 2015)? Without unfolding the debate, it seems clear from this literature that, as usual, the truth lies more in the grey area rather than clearly on one side or the other. Depending on the SES under consideration, the pathogen, the scale of study, and the type of biodiversity indicators, biodiversity can impact health positively or negatively. At this early stage of the field, it would be hazardous to "sell" biodiversity as providing a global ecosystem services for health. It is a possibility (and a number of studies have demonstrated it) but we may rely our reasoning on a biased sample in terms of location, taxonomy or other variables.

These EFGs still need to be developed from a conceptual basis and this should be part of my (our) future work (de Garine-Wichatitsky, Caron & Morand Submitted).

EFG Framework, a preliminary conclusion

Figure 8: EFG framework



The concept of EFG is appealing in theory, as it allows a simplification of the complex mechanisms involving numerous potential players leading to pathogen emergence, while still capturing the role of key players. However, it may be practically challenging because many of the actors potentially involved may be poorly known, or even totally ignored, especially in EID hotspots in tropical regions. Here, our framework allocates potential host populations to appropriate EFGs (Figure 8).

First, researchers and managers must define the limits of the system of interest (Figure 8-top), not only in its biophysical dimensions, but also in its social and economic dimensions, acknowledging the influence of various drivers affecting emergence processes within the EID socio-ecological system (Janes et al. 2012; Wood et al. 2012). This step largely depends on the target population chosen, for instance a human population living within a protected area, livestock population kept in contact with wildlife, or endangered wildlife in contact with livestock.

Second, two independent sources of information can be used to group host populations according to the ecological features or parasites that they share. Interacting host species may be grouped using existing knowledge on their ecological traits, distribution and interactions within the EID ecosystem (Figure 8-middle-left). This step will depend on the modes of transmission of the pathogen/group of pathogens of interest (e.g. direct contact, water-borne, vector-borne) and on existing information regarding the distribution of hosts and associated contact patterns (e.g. habitat use, resource selection, seasonal movements) and their trophic positions (e.g. predation, competition for resources). Another approach consists in using data pertaining to past parasite transmission events in order to identify the most likely future transmission pathways (Caron, Morand & de Garine-Wichatitsky 2012). Host species within an ecosystem may thus be grouped according to the number of parasite species that they share ("shared community of pathogens"), for a given mode of transmission (Figure 8–middle right). The final step of the framework consists in allocating the host species interacting within the SES of interest into EFGs according to the chosen target host, the group of pathogens of concern and available epidemiological and ecological knowledge. This exercise should be viewed as dynamic, because the same host populations might play different roles according to the conditions prevailing under various driving factors (e.g. change in wildlife densities, vaccination), and iterative, as comparing the EFG diagrams generated under different conditions will highlight potential key host populations and mechanisms, which might lead to testable hypothesis.

With no doubt, there will be some debate between experts regarding the allocation of individual host populations in a given EFG or another, and probably also regarding the definition of some EFGs in particular cases. This is good, because the proposed framework will help identify knowledge gaps and assist in the formulation of testable hypotheses (see F.1 and the Ebola example). In some cases, precise information on changes in pathogen genetics, host specificity, transmission modes, incidence patterns, or invasion dynamics relative to the pre-emergence situation will be missing. Here, the framework may assist in knowledge gap analysis to reveal critical information that is missing, suggesting where new research is needed. In other cases, applying the EFG framework will highlight uncertainties regarding the functional roles played by host species, which may be challenged using experiments or manipulations of host populations. The ecology of disease emergence is a dynamic process, and recent emergence events have resulted from the onset of a new player in a given system, or from the change in the role played by an existing player. Similarly, the comparison of EFG diagrams established for the same SES before and after a change in driving forces (e.g. change in land-use policy or demographic pressure leading to agricultural encroachment in wildlife habitat) will pin-point key host populations, or key transmission mechanisms, that may lead to disease emergence in the target population.

I therefore believe that the new (redefined) concepts and framework that I (we) have developed belong to fundamental and empirical research. It provides a roadmap to explore a multi-host disease system. It definitely paves the way for my future research on transmission ecology in multi-host systems in African SES. In the following two chapters I will unfold the main research results I have obtained on two models during the last 10 years while the next chapter will describe briefly a few additional works that I have been developing or collaborating with in relation to the EFG concept.

D. Avian influenza ecology at the wild/domestic bird interface

When I first got involved with a Cirad team from the then ECONAP research unit into Avian Influenza research during the second semester of 2005, the H5N1 HPAI (Highly Pathogenic Avian Influenza virus) outbreak had just spread from Asia to Europe, was about to spread in the Middle East and Africa and the panzootie was threatening of becoming an epidemic and potentially a pandemic (Kilpatrick et al. 2006). At the time, there was little or no information about the ecology of LPAI in wild birds in Africa except for some information in South Africa (Abolnik et al. 2004). Firstly, the FAO asked us to estimate the probability that Palearctic wild birds (birds migrating from Africa to Europe and Asia back and forth) would come back from their winter migration in Africa to Europe with H5N1 HPAI. After this project between 2005-2007, the French Ministry of Foreign Affairs (MAE) asked us through the FSP GRIPAVI project to investigate LPAI and HPAI ecology in 6 study sites, 5 in Africa and 1 in Asia. I was responsible of the site in Zimbabwe and part of the core group of 3 (Nicolas Gaidet - P.I., Julien Cappelle and myself) coordinating the wild bird studies within this project. This resulted in a large dataset that was not only combined across the GRIPAVI project dataset but also with the SA-GAINS project (South African Component of the Global Avian Influenza Network for Surveillance, USAID) implementing research on avian influenza in wild birds in southern Africa (coordinated by University of Cap Town). Altogether, this Zimbabwean and regional data contributed to my PhD thesis (Caron 2011; Caron et al. 2012a; Chiweshe & Caron 2012). The GRIPAVI project ended in 2011 and since then we have been producing a substantial amount of literature on LPAI in wild birds and at the wild/domestic bird interface (the later being my main contribution). I present in the following sections our main results on this model within the disease ecology framework as I presented previously.

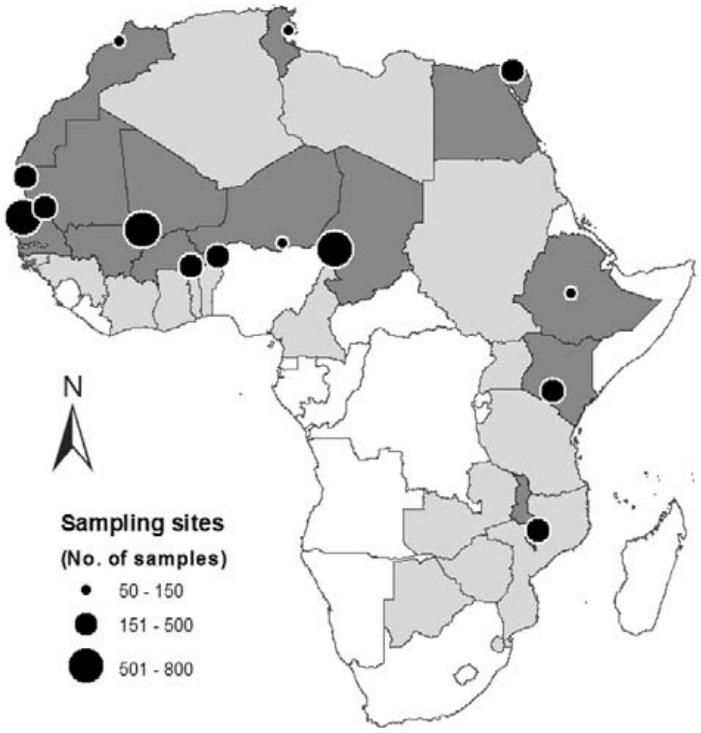
1. Palearctic and Afro-tropical birds are infected by LPAI in Africa

This first result was rather descriptive but required a lot of investment. In 1 year (the FAO project lasted 2 years but most of the sampling activities occurred during the first year), the Cirad team sampled thousands of wild birds in a wide variety of ecosystems in Africa. I was personally in charge of sampling in Niger (a few weeks after the H5N1 HPAI outbreaks), in Chad (in 2006 & 2007), in Mali, in Zambia and involved in training activities for local personal including wild bird captures in Ukraine, South Africa and Malawi (Figure 9).

Our main conclusions were:

- No HPAI H5N1 were detected in the wild birds sampled.
- LPAI were detected and isolated in several wild bird species from several major wetlands of northern, western and eastern Africa, indicating that environmental conditions in Afro-tropical ecosystems are favourable for the persistence and transmission of AIV. Amongst the general scientific opinion, it was previously hypothesised that African environmental conditions (including high temperatures, dryness) would preclude LPAI circulation in African ecosystems.
- LPAI were detected and isolated from Eurasian and Afro-tropical bird species (the later remaining throughout the year in Africa) and during the fall and the spring migrations.

Figure 9: FAO project sampling sites in Africa



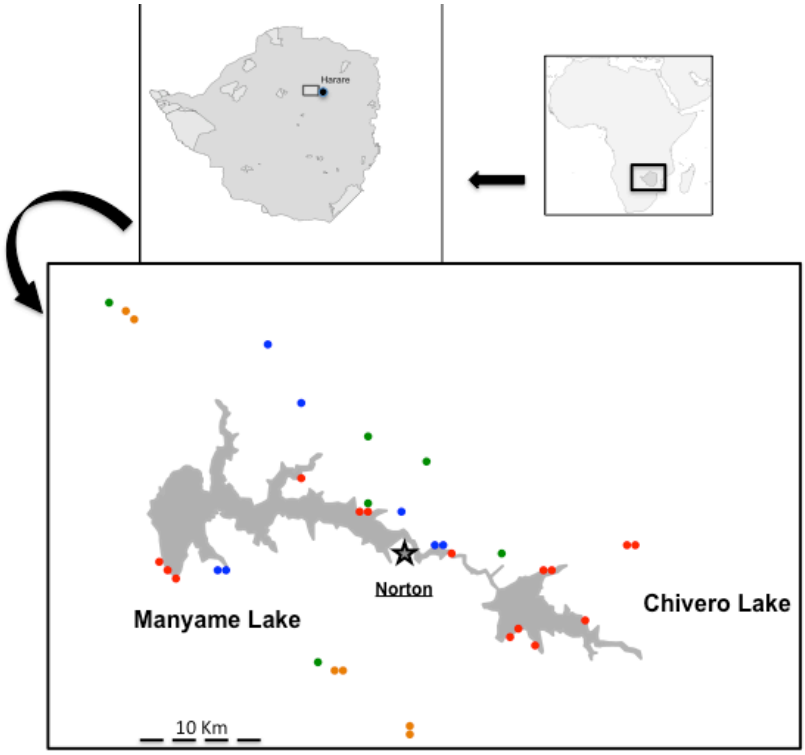
From (Gaidet *et al.* 2007a)

Those results led us to hypothesise the persistence and/or maintenance of LPAI in African bird in Africa. Alternatively, Eurasian (Palearctic) migrating populations could re-introduce yearly new strains in Africa where they would progressively fade away because of the lack of adequate solutions to be maintained in African ecosystems (Gaidet et al. 2007a; Gaidet et al. 2007b). Both hypotheses were not exclusive.

2. LPAI circulates yearlong in wild birds in Africa

In order to test the hypothesis of persistence and maintenance of LPAI in wild birds in African ecosystems, I needed a framework to implement long-term surveillance of LPAI in multi-host systems. From the beginning, it was decided not to simply sample wild birds regularly in the same ecosystem but to combine ecological and epidemiological data. We implemented this in the Mali and Zimbabwean sites and to some extent in the Mauritanian sites. Therefore, in parallel to our sampling scheme, we implemented regular counting sessions in the wetlands studied. Hence we were able to compare the existing wild bird community with the biased sample obtained through capture techniques and therefore the biased in epidemiological data obtained at the wild bird community level (Figure 10 & 11)(Caron et al. 2009).

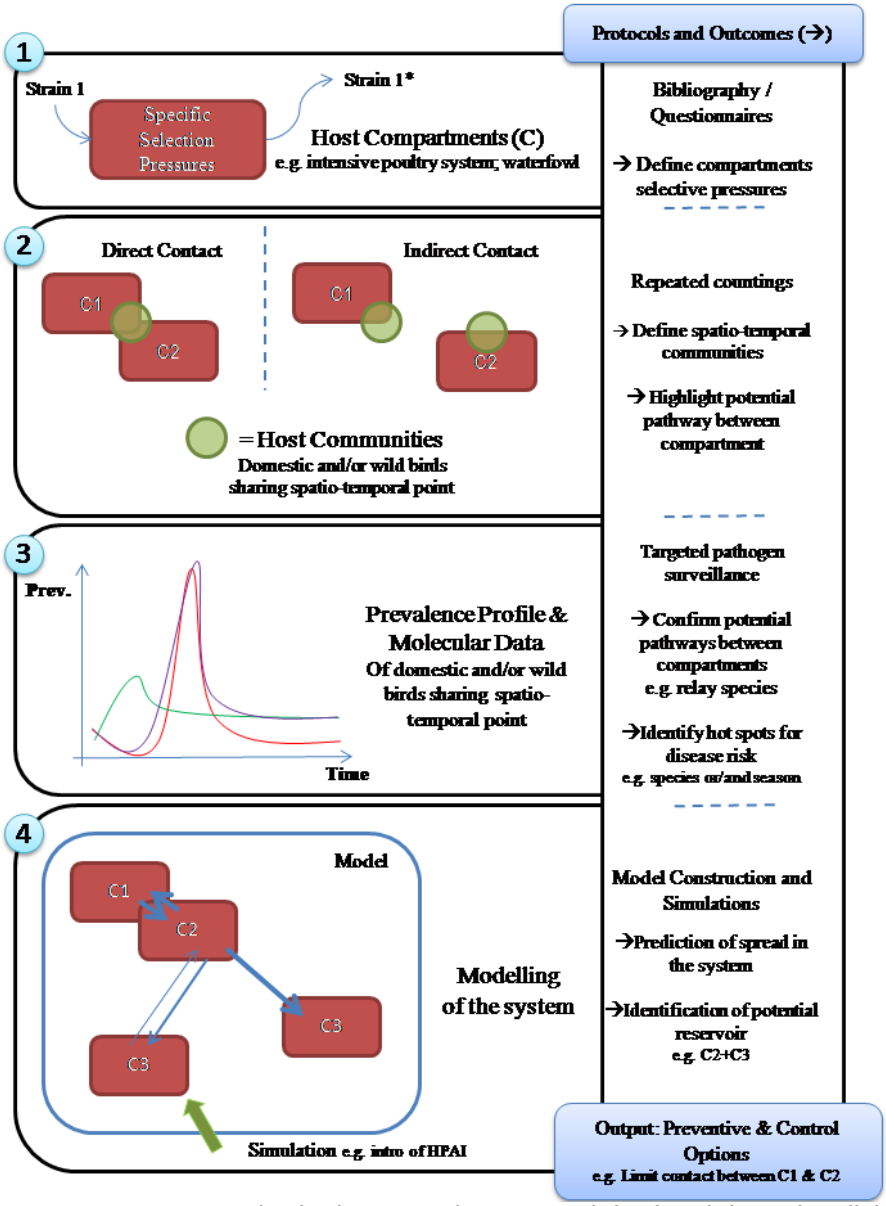
Figure 10: Study site in Zimbabwe



Top right is a map of Africa indicating the location of Zimbabwe. Top left is a map of Zimbabwe indicating the location of the study site (in the rectangle); The bottom section is a map of the study site with on the left, Lake Manyame and on the right Lake Chivero with the main town Norton indicated on the Manyame shores. Red dots indicate waterfowl counting sites; blue dots indicate intensive poultry counting sites; green dots indicate backyard poultry counting sites; yellow dots indicate ostrich farm counting sites (Caron *et al.* 2014).

My results did demonstrate as mentioned in section C.3 "Maintenance vs. persistence" and Figure 5 a persistence of LPAI in wild birds in the lakes Chivero and Manyame (Caron et al. 2011). Our waterfowl community was dominated by Anseriformes (ducks) populations, mainly represented by two species: red-billed teal (*Anas erythrorhyncha*) and the white-faced whistling duck (*Dendrocygna viduata*). Amongst LPAI positive birds, 49.5% were ducks of which 93.7% were dominated by the two duck species mentioned previously. Anseriformes populations were clearly driving the LPAI prevalence in the ecosystem. However, a few Palearctic migrants were positive for LPAI and the LPAI prevalence was higher during the period when Palearctic migrants were present in the ecosystem.

Figure 11: Conceptual research framework for AIV ecology in multi-host systems

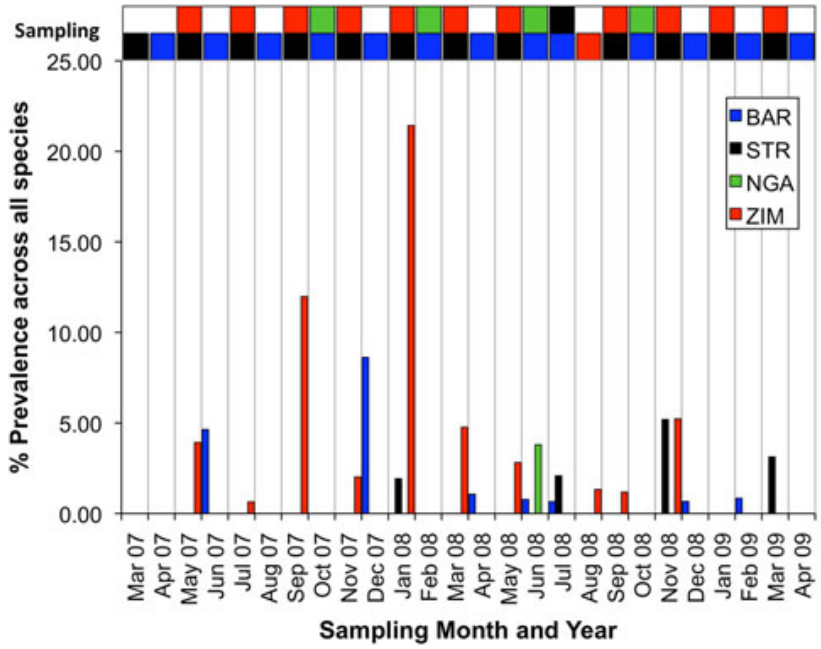


Four steps are presented referring to numbers on top left of each box: 1) Definition of domestic and wild compartments in the ecosystem; 2) Definition of the communities in the ecosystems (temporal and spatial variation); 3) Targeted surveillance to test hypotheses from (2); strains isolation will confirm or not prediction on host selective pressure on viral evolution per compartment; 4) Modelling of the system for predictive purpose and definition of new hypotheses – feedback to (2) and/or (3). On the right vertical box, protocols and outcomes (preceded by arrow) proposed for each box. As an example we could consider: C1= backyard poultry compartment; C2=waterfowl compartment; C3= Intensive poultry compartment (Caron *et al.* 2009).

Finally the second year, we could not detect any LPAI during the rainy season but with only a small sample size (due to the difficulty to catch birds at this season because they disperse to reproduce). I therefore could not distinguish between persistence and maintenance. I also observed the co-circulation of several LPAI strains for two years suggesting that some strains were maintained throughout the year. However the absence of isolation of strains prevent us to go further in the phylogenetic analyses.

These results were confirmed with results from other ecosystems in the related projects. First Cappelle et al. (2012) did indicate persistence of LPAI in wild birds in the Inner Delta of Niger in Mali. On a larger dataset of southern Africa, we also had a clear signal of persistence of LPAI in 6 different ecosystems (Cumming et al. 2011)(Figure 12). Once these preliminary results were obtained the next step was to try to explore which factors could drive the dynamics of LPAI in wild birds.

Figure 12: Prevalence of AIV in southern Africa in 4 ecosystems



Prevalence of avian influenza by site and month across all captured birds. Sites are BAR Barberspan (South Africa), STR Strandfontein (South Africa), NGA Ngami (Botswana) and ZIM Zimbabwe (Manyame and Chivero). Note that (1) another 294 birds were sampled in Mozambique over the same period, with no AIV positives found; and (2) BAR, STR and ZIM were sampled every 2 months and NGA every 4 months, so birds were not sampled in some months. The shaded squares at the top of the chart indicate when a given site was sampled, using the same colour codes as the bars (Cumming *et al.* 2011).

3. Drivers of LPAI dynamics in wild birds

In the northern hemisphere, the seasonal dynamics of AIV transmission have been related to two main ecological factors: i) the recruitment of immunologically naïve young birds into the host populations; ii) the seasonal aggregation of birds at pre-migration sites in late summer (Munster et al. 2007). These two factors acting synchronously have been proposed to explain the seasonal peak of AIV infection observed in late summer and autumn in boreal and temperate regions of the northern hemisphere (Krauss et al. 2004; Munster et al. 2007). AIV prevalence in waterfowl can increase up to 30% with the influx of juveniles in the post-breeding period and pre-migration staging as a consequence of reproduction synchrony (Hinshaw & Webster 1980). This peak in prevalence has mostly been attributed to juvenile immune system naivety, as well as the effect of host density on contact rate (Gunnarsson et al. 2012). Environmental factors such as water temperature, salinity and pH influence the survival of AIV in water and therefore environmental transmission (Stallknecht et al. 1990; Roche et al. 2014). AIV dynamics are

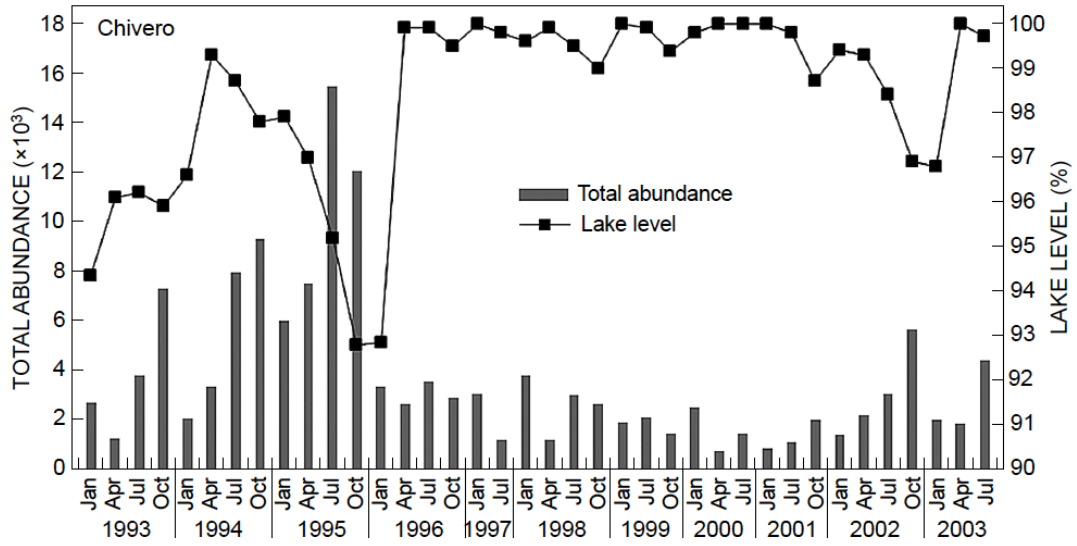
therefore strongly dependent on host density, host population immunity and survival of the virus in the environment (Mundava *et al.* Submitted).

The datasets available offered the opportunity to test the influence of these drivers on LPAI dynamics in wild birds at the ecosystem, regional and continental level. Josphine Mundava, the PhD student that I co-supervised through the GRIPAVI project, addressed the ecosystem level. I led a study at the regional level. And collectively, we addressed the continental level with a coordination by Nicolas Gaidet who led both continental-level studies.

Ecosystem level

I manage to secure the access to a bird count dataset of 10 years implemented by the same dedicated ornithologists over the years and managed to have them resume their protocol for another 2 years. Josphine analysed the relationship between environmental drivers (e.g. rainfall, lake level) and the waterfowl community composition (Mundava et al. 2012).

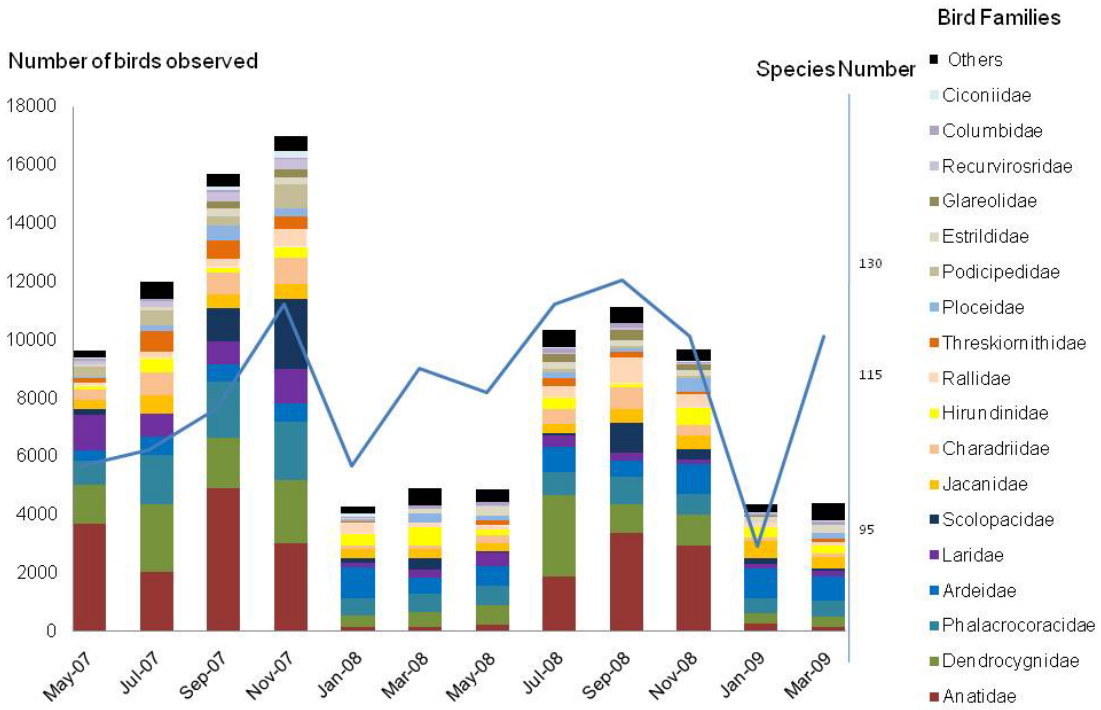
Figure 13: Variation in waterfowl abundance and lake level in Chivero Lake (1993-2003)



(Mundava et al. 2012)

Overall, a clear relationship was observed in lakes levels and the waterfowl global abundance on their shores (Figure 13). This relationship is explained by a classic link between resource availability and attraction of consumers. As the Lakes' shores recede due to the dry season and the absence of rains, it exposes large areas of good grazing quality in a landscape where these resources are scarce during this season. The lakes' shores offer therefore a refuge for waterfowl. During the rainy season, as resources are widely available in the landscape and non-perennial water bodies numerous, birds disperse. From an epidemiological perspective, this means that dry seasons are at risk of pathogen transmission when numerous species congregate on small areas and that rainy seasons are less at risk because of lower host densities. In our recent dataset, these relationship was still present (Caron et al. 2010)(Figure 14).

Figure 14: Waterfowl abundance and lake level in Chivero and Manyame during two years

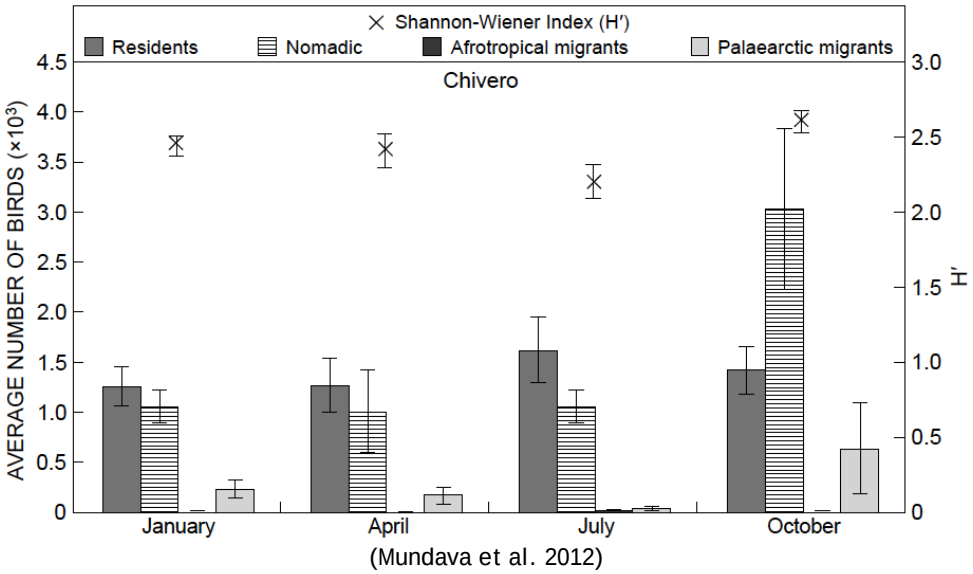


(Caron et al. 2010)

More recently, the composition of the waterfowl community also varied according to season as can be seen on Figure 14 when species are classified according to bird families but also on Figure 15 when species are classified according to their migratory behaviour. Nomadic birds mostly duck species (the two species previously mentioned) contribute mainly to the higher waterbird abundance during the dry season. These species respond regionally to the abundance of resources and therefore their behaviour is difficult to predict at an ecosystem scale as can be seen by the difference between the two years in Figure 14 (Dodman & Diagana 2007). The second important observation from these results is that Palearctic migrants arrive around September-October in the ecosystem and this is when we expect to see a rise in AIV prevalence if those species play a role in introducing LPAI strains in the ecosystem annually.

As nomadic duck species are mainly Anseriformes (in abundance) and therefore potentially maintenance hosts for LPAI, we observe a higher risk of AIV circulation in the ecosystem during the dry season but a non-zero risk of circulation all year long. The risk of AIV introduction in the ecosystem is higher also during the dry season. Our AIV data (Figure 5) did corroborate those hypotheses. However the low proportion of Paleartic migrants sampled and tested prevented more insights into their role in AIV ecology in this ecosystem.

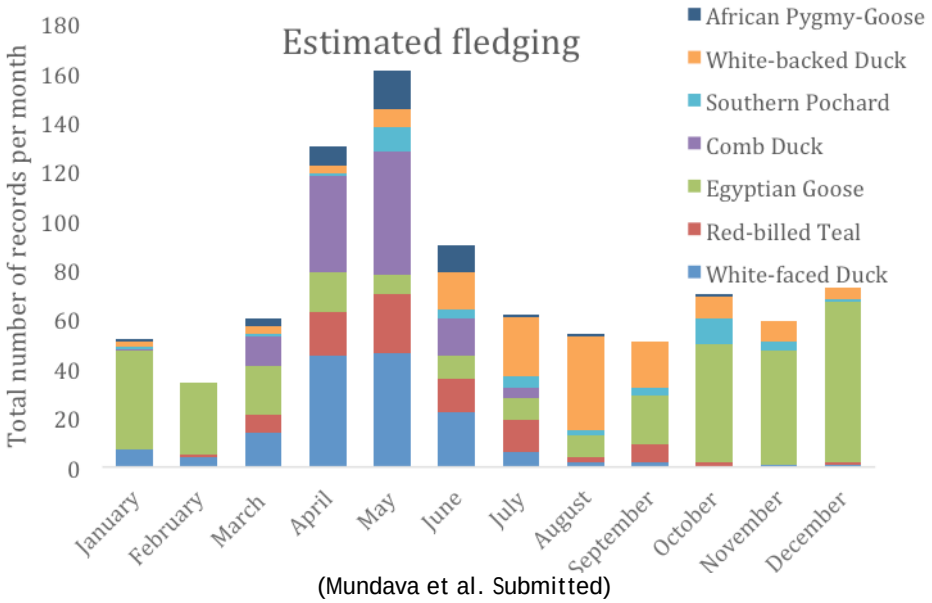
Figure 15: Interseasonal variation in the waterbird community and Shannon-Wiener diversity index (H') for Lake Chivero



We then decided to test the hypothesis of the influence of the reproduction phenology (Mundava *et al.* Submitted). First, we used a large dataset of waterfowl breeding records collected across Zimbabwe over a century to describe the seasonal breeding patterns for the most common waterfowl species. Second we used bird count data collected at a breeding site (i.e. Hwange National Park) and at a post-reproduction aggregation site (i.e. Manyame catchment) to describe the seasonal dynamics in waterfowl local abundance. We used these two data sources to test the hypothesis that there is a shift in the local abundance in waterfowl between the two sites as birds move from the breeding area to the aggregation site.

Although breeding seasonality was observed at the species level, it was less pronounced than the seasonality described in the temperate and boreal regions, with breeding period for each species in Zimbabwe spanning over seven to twelve months (Figure 16). Second, we identified a general asynchrony in the seasonality of breeding periods among species, in contrast to the seasonal pulse in juvenile production observed in the temperate and boreal regions. The extended and asynchronous breeding periods among waterfowl species probably result in a continuous year-round influx of susceptible individuals in the waterfowl community. Third, we identified two dominant waterfowl species (i.e. White-faced Duck and Red-billed Teal), in terms of abundance, in the waterfowl community associated with an observed peak in juvenile numbers during the cold-dry season at the aggregation site. Fourth, we found no significant relationship between the production of juveniles and the seasonal variation in AIV prevalence, but a significant relationship between the seasonal variation in the local abundance of waterfowl and the seasonal variation in AIV prevalence.

Figure 16: Seasonal variation in estimated cumulative fledging period for all duck species with sufficient breeding data



(Mundava et al. Submitted)

From a disease ecology perspective, we suggest that the drivers of AIV prevalence identified in the northern hemisphere (presence of susceptible juveniles and seasonal aggregation) are present in the Afro-tropical regions but their pace and impact are not as pronounced as in the temperate and boreal regions (Table 6). First, the recruitment of juveniles into the waterfowl population is gradual, with species producing young at different times of the year (breeding asynchrony) in comparison to the synchronised reproduction in the temperate and boreal regions (Hinshaw & Webster 1980; Fouque et al. 2004). The slow turnover of susceptible young over time suggests a corresponding gradual pace of infection resulting in the lack of significant relationship with the seasonal variation in AIV prevalence. Furthermore, there is a clear movement of juveniles (among the most abundant species) and adults from breeding sites to the aggregating sites, and we would expect a rise in AIV prevalence with the introduction of immunologically naïve young into the population. However, we suggest that the period of contact between juveniles and adults is too brief for pathogen transmission, as during that time, the adult birds at the aggregating sites would be moving away from the main flocks to move to moulting sites. This suggestion is supported by our observation of more than 75% of White-faced Duck and Red-billed Teal juveniles in the Manyame catchment in the month of July, with very few adults observed. However, this hypothesis is largely speculated.

Table 6: Comparison of ecological drivers associated with the seasonality of AIV prevalence in wild ducks in temperate-boreal and Afro-tropical regions

		Afro-tropical regions			Temperate and boreal regions			
Variable	Proxy	Hot-Wet	Cold-Dry	Hot-dry	Winter	Spring	Summer	Autumn
Inter-host contact	Host density	*	*	**	***	*	**	***
Proportion	Proportion	*	**	*	0	0	**	***

of susceptible hosts	of juveniles							
Environmental transmission	Temperature	*	**	*	***	**	*	**
AIV prevalence	Prevalence estimation	*	*	*	0	0	**	***

The Afro-tropical regions column is derived from the results of this study. The boreal or temperate regions of the Northern Hemisphere column are based on literature (Hinshaw & Webster 1980; Fouchier et al. 2007; van Dijk et al. 2014). The number of * depicts an increased force of the factor under scrutiny (Mundava et al. Submitted).

The process of aggregation in this Afro-tropical region occurs similarly as to the temperate and boreal regions. However, unlike in the temperate and boreal regions where pre-migration staging results in abrupt aggregation events (Munster et al. 2007; van Dijk et al. 2014), the process in the Afro-tropical region is not as pronounced as waterfowl mostly aggregate as a response to resource availability as permanent wetlands act as refuges in the dry seasons (Mundava et al. 2012). The pace and magnitude of aggregation is therefore dependent on the status of the wetlands – which is mostly dependent on the rainfall patterns of the previous wet season. Therefore there is a gradual and unpredictable pace of density-dependent infection in the Afro-tropical regions as aggregation is not as abrupt as in the temperate and boreal regions, and this would result the lack of clear seasonal patterns in AIV prevalence, or a peak in AIV prevalence at times of high waterfowl density (Gunnarsson et al. 2012).

The asynchrony and its timing observed in the breeding phenology of ducks could play a role in the year-long persistence of AIV in waterfowl communities in the Afro-tropical regions, already suggested in previous studies in association with environmental factors (Caron et al. 2011; Cumming et al. 2011; Cappelle et al. 2012). These differences imply a variation in the risk of AIV circulation in relation to domestic poultry systems and a need to adapt to these variations when drafting and implementing protection measures. Further research should focus on profiling the role of environmental factors in the persistence of AIV (Nazir et al. 2010) within waterfowl populations, and identifying the types and dynamics of AIV strains circulating in relation to host communities.

Regional level

Our study so far concentrated on Anseriformes populations at the ecosystem level. Although numerous studies of LPAI in waterfowl and wild birds have been published, encompassing tens of thousands of sampled wild birds, we still know relatively little about the susceptibility of individual bird species to AIV in relation to the global number of bird species (Olsen et al. 2006). The avian community in a single ecosystem can include hundreds of interacting species. In addition, the response of bird species to specific AIV subtypes (16 hemagglutinins and 9 neuramidases known) is variable and prevalence patterns of specific subtypes will be determined by the bird cenosis. In their synthesis of wild bird low pathogenic avian influenza surveillance worldwide, Olsen et al. (2006) found that out of more than 90 000 birds sampled, 54% were Anseriformes and 25% Charadriiformes. As a consequence, little information on AIV prevalence in the rest of the avian community has been published, and much of what has been published has been obtained as “by-catch” from capture protocols that have been focused on ducks.

The minimum sample sizes that would be necessary to confidently estimate prevalence for most non-target bird species are often not reached, with the risk that the common practice of focusing mainly on Anseriformes may be overlooking the role of other bird groups in the epidemiology of AIV in waterfowl communities.

Biases in the selection of species to sample are not the only problem in available data sets for AIV. The comparison between sample and community composition is a fundamental parameter in epidemiological studies (Dohoo, Martin & Stryhn 2009). In many cases, a lack of information regarding the composition of the wild bird community from which the sample is taken makes conclusions from AIV studies even harder to interpret. A total of 100 positive samples from mallard ducks (Anas platyrhynchos), for example, carries quite different epidemiological implications if mallards represent 0.1% versus 90% of the number of wild birds present in the ecosystem; and similarly, the relevance of 100 positive samples from mallards differs if the system contains 10 or 100 other species. Interpretation of the role of a species in pathogen maintenance cannot be done rigorously without considering the potential role of the rest of the community. The sampling bias that is attendant on any field captures of wild birds should therefore be a crucial parameter in wildlife epidemiological studies.

I therefore applied our epidemiological functional group approach to the southern Africa regional dataset gathered under GRIPAVI and GAINS-SA. The idea underlying functional group analysis is that broad, community-level trends in processes of interest can be detected by replacing a taxonomic classification of hosts with a classification that groups hosts according to their functional role in the epidemiology of a pathogen or a group of pathogens (Caron, Morand & de Garine-Wichatitsky 2012). Although 'AIV' describes a group of pathogens, we treat it as a single pathogen, ignoring AIV subtype variability, because the sparse information available does not suggest that AIV modes of transmission significantly differ among subtypes (see (Caron et al. 2011; Cumming et al. 2011) for details). Hosts in an EFG share a common function in the epidemiology of the pathogen(s) of interest. We used the concept of EFGs to (1) investigate the ecology of AIVs in three different wild bird communities in southern Africa; and (2) critique the current scientific paradigm for field investigations of AIV in wild birds.

We used the wild bird census dataset to first allocate species to EFGs according to two epidemiological functions (maintenance vs. non-maintenance species, and the potential to introduce AIV strains through migratory behaviour; note that the functional groups, as described later, differ from the functions themselves) according to current AIV epidemiology dogma (Table 7). We ranked each group in relation to their expected contribution to pathogen prevalence and then used our rankings to calculate relative a priori risk for each group. We then compared these a priori relative risks (which are effectively predictions, generated by accepted knowledge) to our empirical data on observed prevalence per group. In other words, we used EFGs as a way of exploring the degree to which empirical data match commonly held assumptions, rather than adopting the commoner approach of attempting to classify species into EFGs based on our own data. Our results suggest that commonly held assumptions might require some re-thinking.

Table 7: Epidemiological functional group and relative risk

EF2 / EF1	Anseriforms *Ans=3*	Charadriiforms *Cha=2*	Rest of Community *RoC=1*
Resident *Res=1*	*Ans-Res=3*	*Cha-Res=2*	*RoC-Res=1*
Afro-tropical migrant *Afr=2*	*Ans-Afr=6*	*Cha-Afr=4*	*RoC-Afr=2*
Palaearctic migrant *Pal=3*	*Ans-Pal=9*	*Cha-Pal=6*	*RoC-Pal=3*

Epidemiological functional groups used in this study, based on the two epidemiological functions related to the maintenance and introduction potential respectively of AIV in southern African ecosystems. Numbers represent qualitative estimations of the AIV relative risk for each epidemiological function and for each EFG. For each cell, the qualitative estimation of the relative risk is calculated by multiplying values of the relative risk of EFGs from EF1 and EF2 (Caron *et al.* 2010).

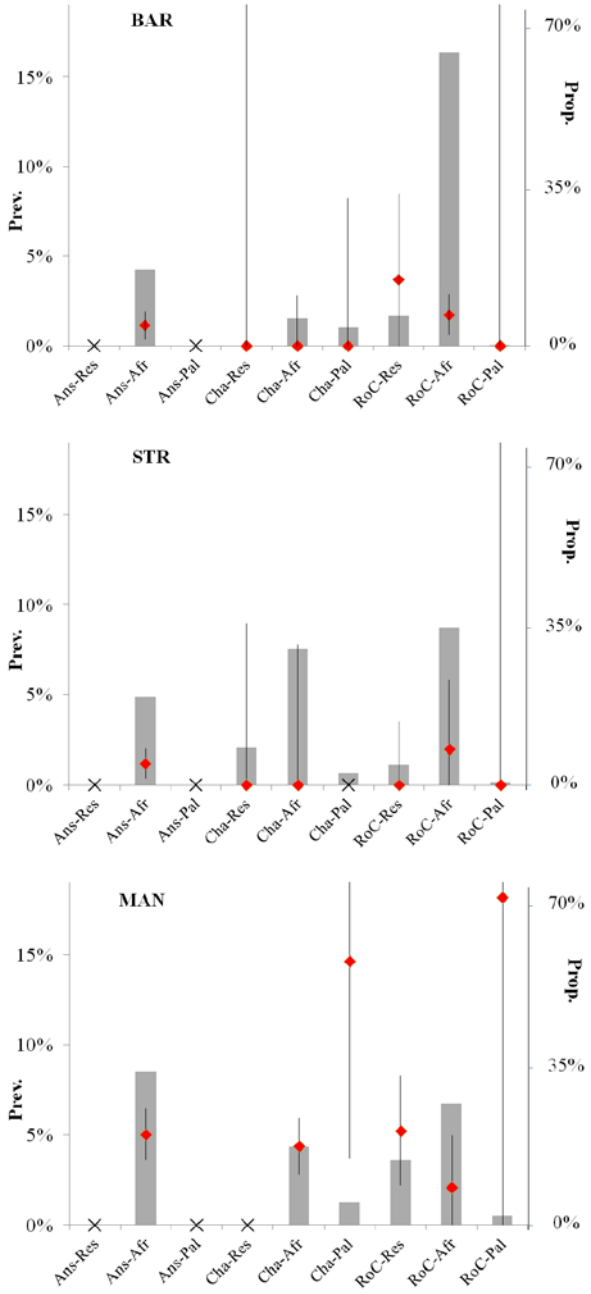
Analysing this epidemiological dataset in accordance with the current dogma of AIV in wild birds, Anseriformes prevalence appears to drive prevalence at the community level in each ecosystem (Caron et al. 2011; Cumming et al. 2011). By including bird community data and the composition of the captured sample, and taking into account the EFG approach, we obtain a different perspective (Figure 17): (1) different bird communities predict different AIV risks ("a priori risk") using the EFG approach in the three ecosystems, a result validated by the "estimated risk" using field AIV prevalence; (2) sampling bias can explain discrepancies between the "a priori risk" and "estimated risk" for AIV prevalence; and (3) Anseriformes play an important role in AIV epidemiology in waterfowl in the three ecosystems, as assumed by the current understanding of AIV ecology in wild birds, but other bird groups identified at the EFG level show unexpectedly high prevalence, and could play a role in the local epidemiology of AIV.

The EFG approach thus appears to be successful in improving our understanding of the role of wild birds in the epidemiology of AIV by highlighting potential epidemiological functions for unconsidered bird groups, identifying gaps in knowledge or sampling and suggesting new hypotheses. The EFG approach also carries the benefit of making better use of "by-catch" samples, which are often portrayed in AIV studies as secondary-level data (Hansbro 2010).

Current knowledge about global AIV epidemiology applies to some extent to AIV epidemiology in these three southern African ecosystems. Notably, the important role of Afro-tropical Anseriformes in the epidemiology of AIV is confirmed in southern Africa (Caron et al. 2011; Cumming et al. 2011). However, higher than predicted AIV prevalence in other bird groups challenges the hegemony of Anseriformes as the primary actor in the maintenance of AIV in these ecosystems. Firstly, the AIV prevalence estimated in the resident RoC group (Rest of the Community after removing Anseriformes and Charadriiformes) in BAR (Barberspan ecosystem, South Africa) and MAN (Manyme ecosystem, Zimbabwe) is not significantly different from the prevalence in the afro-tropical Anseriformes group across the two years of the study. The same observation can be made for afro-tropical Charadriiformes and to a lesser extent for the Palaearctic RoC group (with a high prevalence but a small sample size leading to a high maximum undetectable prevalence) at MAN. For all three ecosystems, the majority of AIV infected birds would not belong to the afro-tropical Anseriformes group, contrary to what might be expected for the reservoir of the disease. In BAR and STR (Strand Fontein ecosystem, South Africa), for example, the majority of the infected birds would

belong to the afro-tropical RoC group. Finally, all EFGs but one that represent more than 15% of the community have a detectable prevalence, suggesting a frequency-dependent role of EFGs in relation to AIV prevalence.

Figure 17: AIV prevalence for each EFG in relation to bird community composition in the three sites



For each site (BAR - South Africa, STR - South Africa, MAN - Zimbabwe): a) prevalence and points with 95% confidence interval (left axis) for each combination between EF1 & EF2 (Ans= Anseriformes, Cha= Charadriiformes, RoC=Rest of Community, Res=resident, Afr =afro-tropical, Pal=Palaearctic migrant); b) Proportion of each bird group in the bird community observed and bars (or counted) during the 2 years of the project (right axis)(Caron *et al.* 2012b).

A role for these EFGs in the persistence or maintenance of AIV in our study ecosystems cannot be ruled out, even though this conclusion would go against current dogma surrounding AIV epidemiology in wild birds (Stallknecht & Brown 2007). Most studies of AIV ecology in wild birds have been implemented in temperate climates (Olsen et al. 2006). The current dogma in this field therefore comes from studies implemented in specific biophysical conditions (e.g. climatic condition, ornithological diversity) that have been shown to influence the epidemiology of AIV (Rohani et al. 2009). Often, this dogma is taken for granted when studies are implemented in other regions with different biophysical conditions. Our results indicate that more attention should be given to designing local hypotheses in reference to global assumptions: some space should be left for alternative hypotheses and the inclusion of count data and the sampling of other bird species than Anseriformes and Charadriiformes will serve this purpose.

Our data support the idea that some EFGs play an important role in the persistence and/or maintenance of AIV in southern African ecosystem. They also imply that comparisons of prevalence data from multiple sites (even if the sampling was done at similar time) are compromised if environmental and ecological variability is not accounted for.

This study helped us challenging the current AIV ecology in wild bird dogma that led us to design the ideas in section D.5.

Continental level

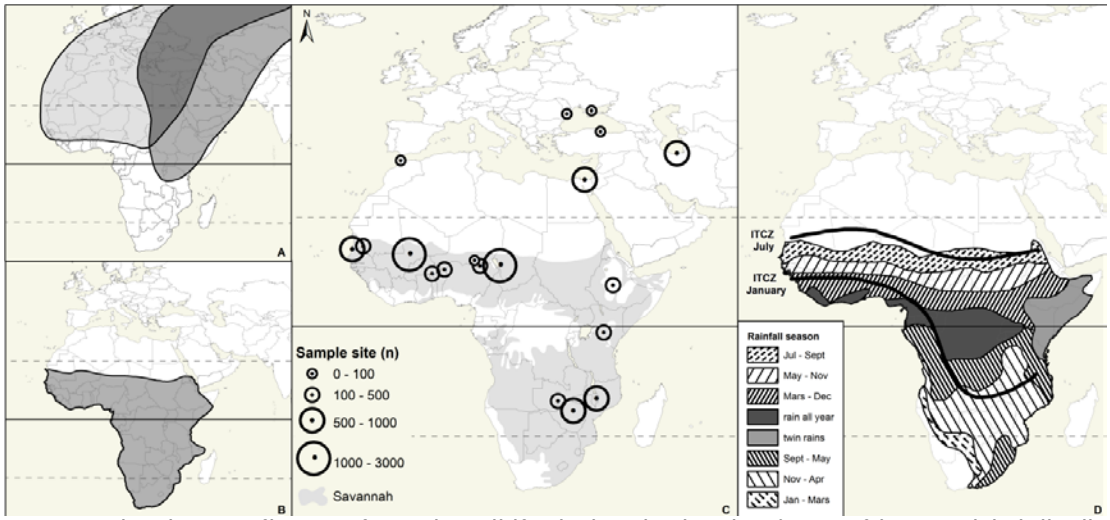
Despite considerable effort for surveillance of wild birds for avian influenza viruses (AIVs), empirical investigations of ecological drivers of AIV prevalence in wild birds are still scarce. Our research efforts on AIV ecology in wild birds in Africa put us in a position to do a continental scale analysis to test hypotheses based on current understanding of AIV ecology in wild birds in the Northern hemisphere. This led to two studies, on investigating the role of Anseriformes and the other the role of Charadriiformes in AIV ecology and the drivers of AIV in these populations of bird orders in Afro-tropical regions (Gaidet et al. 2012a; Gaidet et al. 2012b).

In the first study, we used a continental-scale dataset (Figure 18), collected in tropical wetlands of 15 African countries, to test the relative roles of a range of ecological factors on patterns of AIV prevalence in wildfowl (Gaidet et al. 2012a). We used generalized linear mixed models and a model comparison approach to assess the ability of various ecological factors to explain species, seasonal and geographical variations in AIV prevalence measured in wildfowl across Afro-tropical regions. We tested factors (Table 9) related to i) the probability for a wildfowl species to be infected, including its migratory behaviour, foraging behaviour and taxonomic group, and ii) the capacity of the local host community and environment to perpetuate and transmit the virus, including the host density (at species and wildfowl community level), the proportion of the potentially most competent species (Eurasian, dabbling or *Anas* species) in the wildfowl community, the climate (temperature and aridity indices), as well as the timing relative to Eurasian migration and to congregation during the dry season.

Our results indicate that variations in AIV prevalence in wildfowl at a continental scale were related to several host ecological factors operating at both species and community level, including the species taxonomic group, the local

density of the wildfowl community and the season when Eurasian migratory birds winter in Africa. The timing relative to the dry season congregations, the composition of the local wildfowl community and the climatic variables were relatively poor predictors of AIV prevalence. It also appears that sampling the respiratory tract may be as important as sampling the digestive tract to detect AIV infection in wildfowl.

Figure 18: Presentation of the study design



A. Two main migratory flyways of Eurasian wildfowl wintering in sub-Saharan Africa; B. Global distribution range of Afro-tropical wildfowl over the African continent ; C. Location of wildfowl sampling site for detection of AIV presented in our study; D. The timing and duration of rainfall in sub-Saharan Africa adapted from Jones (1995), the thick lines show the seasonal movement in the position of the Inter-Tropical Convergence Zone (ITCZ). Map produced by M. Gély©cirad (Gaidet *et al.* 2012a).

Prevalence was positively related to the density of wildfowl measured at the community rather than at the species level, suggesting aggregation of infection through interspecies mixing (Figure 19). The density of the wildfowl community varied widely between sites and seasons, in relation to the seasonal variations in wetlands surface and the massive flux of Eurasian migratory wildfowl but also Afro-tropical wildfowl congregating at permanent wetlands during the dry season or, conversely, with the dispersal of birds to newly flooded wetlands after the onset of the wet season. The proportion of Eurasian wildfowl in the wildfowl community was poorly related to the variations in AIV prevalence suggesting that the influx of Eurasian wildfowl influences AIV transmission by increasing the local wildfowl density but that the geographical origin of birds may not matter much. We thus found no support to our initial prediction of a potential difference between Eurasian and Afro-tropical wildfowl in previous AIV exposure and susceptibility to re-infection, neither at the community level (proportion of Eurasian species) nor at the species level.

Table 8: Definition of the ecological factors and explanatory variables of the study

Ecological factors	Explanatory variables	Definition
Species traits	Species	Species name
	Anas genus	*Anas* species versus non-*Anas* species
	Foraging behaviour	Mainly dabbling, diving or mixed grazing-dabbling
	Origin	Eurasian migratory or Afro-tropical birds

	Early migrant	Garganey versus other Eurasian Ducks
Local wildfowl abundance	Species abundance	Population size of sampled species
	Eurasian abundance	Population size of all Eurasian wildfowl sp.
	Anas abundance	Population size of all Anas sp.
	Total abundance	Population size of all wildfowl sp.
	Species density	Density of sampled species
	Eurasian density	Density of all Eurasian wildfowl sp.
	Anas density	Density of all Anas sp.
	Total density	Density of all wildfowl sp.
Local climate conditions	Mean annual temp.	Annual mean of daily average temperature (°C)
	Max. annual temp.	Annual mean of daily max. temperature (°C)
	Mean month temp.	Mean of daily average temperature of month of sampling (°C)
	Max. month temp.	Mean of daily max. temperature of month of sampling (°C)
	Mean annual rainfall	Total annual precipitation (mm/year)
	Mean annual PET	Potential evapo-transpiration (mm/year)
	Mean month PET	Potential evapo-transpiration (mm/month)
	Aridity index	Annual rainfall to annual PET ratio
Seasonal Eurasian migration	Timing relative to Arrival of Eur. migrants	No. of days between median sampling date since 1st sept.
Seasonal congregation	Timing relative to End of the dry season	No. of days between median sampling date since the onset of the previous wet season
Southward seasonal migration	Latitude	X coordinates of sampling site
Nuisance parameters	Sampling type	double, single cloacal/fecal, single oropharyngeal swab
	Laboratory	
	Year	

* Abbreviation of variable used in model definition (Gaidet *et al.* 2012a).

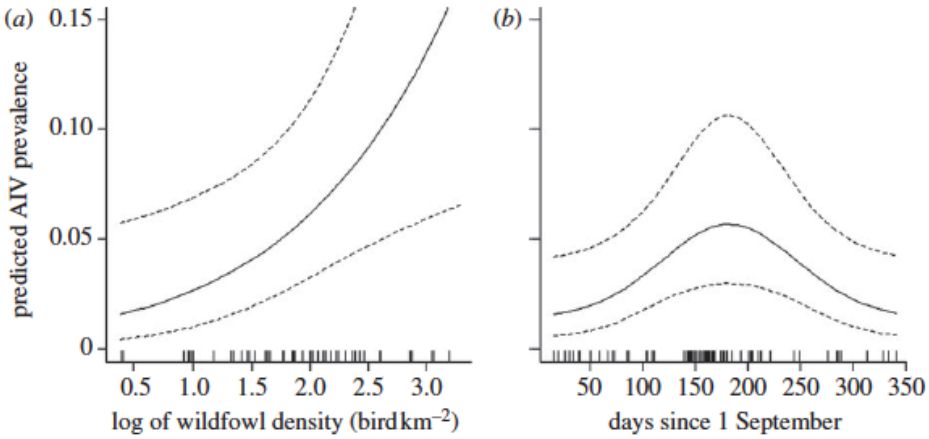
Maximum daily temperatures in most Afro-tropical regions may be over a threshold throughout most of the year where high temperatures prevent the perpetuation of AIV in the environment by more than a few days (Stallknecht et al. 2010). The positive association, which we found between AIV prevalence and the local wildfowl density with no influence of climatic conditions, suggests a predominant role of direct inter-individual transmission via the respiratory route (Kleijn et al. 2010) or via short-lived viruses recently shed in the environment, rather than an indirect transmission via viruses persisting in the environmental reservoir. By contrast, in temperate regions theoretical models of AIV dynamics suggest a greater role for indirect environmental transmission than for density-dependent transmission (Rohani et al. 2009; VanDalen et al. 2010).

Surprisingly, seasonal variations in prevalence were poorly related to the timing of congregation of wildfowl at the end of the dry season. In Palaearctic and Nearctic regions, concentration of wildfowl births into a short seasonal breeding period generates a pulse of immunologically naive birds into the host population as mentioned previously (Altizer et al. 2006; Carver et al. 2009). In Afro-tropical

regions, extended breeding seasons produce a more gradual recruitment rate of juveniles into the host populations as presented in the ecosystem-level study. The seasonal congregation of wildfowl in the dry season in the tropics is also more progressive than the northern migration flocking as it results from the progressive drying of wetlands while migration flocking results from a social gathering behaviour (Mundava et al. 2012). These extended breeding seasons and progressive seasonal congregation may slow down the turnover rate of susceptible birds in the wildfowl community. The seasonality of AIV prevalence in our study was accordingly much less pronounced than in Europe (0–25%) (Wallensten et al. 2007) or North America (0–60%) (Olsen et al. 2006). This should reduce the controlling effect of population immunity on AIV transmission and promote a lower but continuous annual circulation as observed in a southern African wetland (Caron et al. 2011).

Our results indicated that AIV prevalence increases during the period when Eurasian migratory waterbirds (including non-wildfowl species) winter in sub-Saharan Africa and decrease after they migrate back to Eurasia. The arrival of Eurasian migrants constitutes a massive influx of hosts in the local waterbird community but also a potential source of AIV introduction. Eurasian wildfowl are largely absent in the regions south of the equator but large numbers of other Eurasian waterbird species, in particular shorebirds (Charadriiformes), winter in southern Africa. The role of shorebirds in the ecology of AIV is still unclear with highly contrasted results from Nearctic and Palaearctic regions (Krauss et al. 2004; Munster et al. 2007). A low prevalence has been reported globally in non-wildfowl species (less than 2%)(Olsen et al. 2006; Krauss & Webster 2010) suggesting that they play a lesser role in the perpetuation of AIV, though locally shorebirds may have a significant role (Krauss & Webster 2010). Phylogenetic analyses also indicate that inter-continental transfer of AIV genes, though occasional, do occur in shorebirds (Dugan et al. 2008).

Figure 19: Predicted AIV prevalence for Anas species of waterfowl sampled in Afro-tropical regions



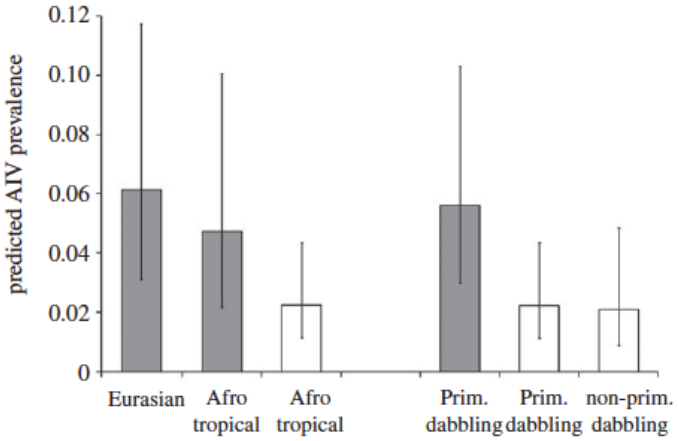
Predicted AIV prevalence (95% CI, dashed lines) for Anas species of wildfowl sampled across Afro-tropical regions in relation to the density of the wildfowl community and the timing relative to the arrival of Eurasian migrants (1 September used as a reference date). Prevalences were estimated for birds tested concurrently for cloacal and oropharyngeal samples, based on the highest rank model, with other predictor variables set to their mean value over the dataset. The distribution of data points is presented as rug plots along the x-axis (a vertical bar for each sampling occasion) (Gaidet *et al.* 2012a).

Difference in prevalence between species was better explained by the taxonomic group than by the foraging or the migratory behaviour of species (Figure 20). Anas species had higher prevalence than non-Anas species even when we account for difference in foraging behaviour or geographical origin of birds. These results support the hypothesis (Munster & Fouchier 2009) that there might be intrinsic differences between wild bird species, including between wildfowl taxonomic groups, in their receptivity to AIV infection. Despite large differences in the proportion of Anas species in the wildfowl community between our study sites and seasons (1–96%), variations in prevalence were poorly related to this variable. This suggests that the absolute rather than the relative number of birds from Anas species may influence AIV transmission.

In our study, the detection rate of AIV was similar in oropharyngeal and in cloacal samples and testing birds for both types of sample produced higher infection rates since birds were rarely found concurrently infected for both types. These results highlight the role of the respiratory tract for the replication of AIV.

Our results provide a unique contribution to our understanding of the ecology of AIV in wild birds in tropical ecosystems but also offer a number of novel insights for understanding the general influence of seasonal fluctuations in animal density and migration on infectious disease dynamics.

Figure 20: Mean AIV prevalence estimated for Anas and non-Anas species



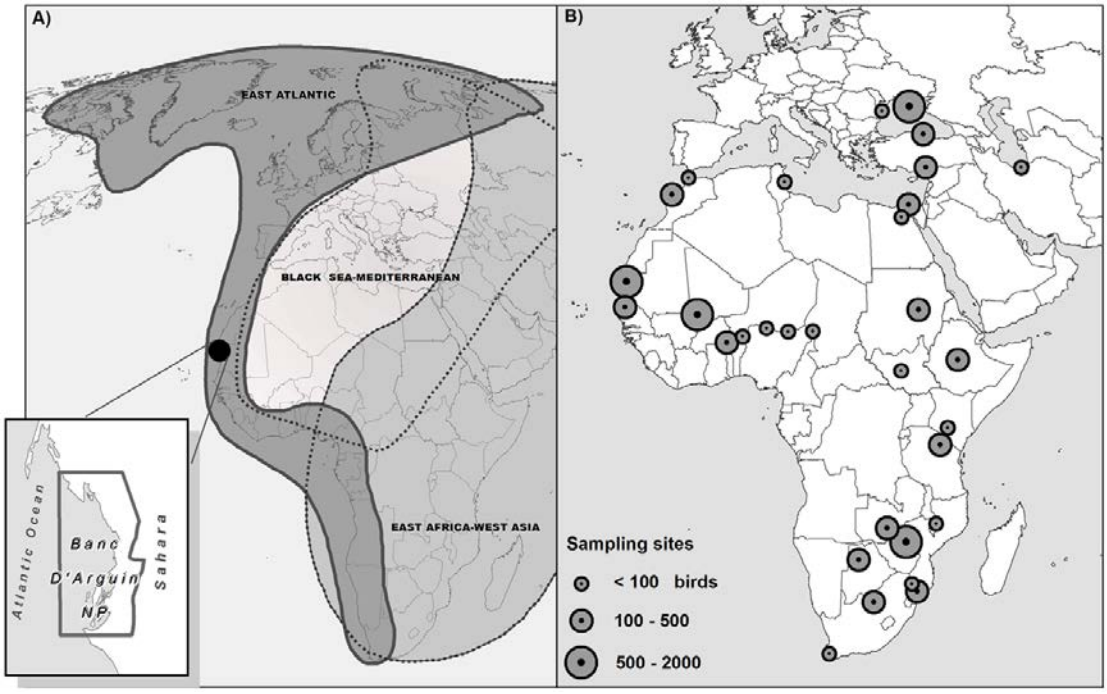
Mean AIV prevalence estimated for Anas (shaded bar) and non-Anas species (unshaded bar) of wildfowl belonging to distinct migratory groups and ecological guilds. Prevalences (95% CI, bars) were estimated for birds tested concurrently for cloacal and oropharyngeal samples, based on the highest rank model (table 3) after substituting the variable Taxonomic group by the composite variables Origin-Taxonomic group or Foraging behaviour-Taxonomic group. Other predictor variables were set to their mean value over the dataset (Gaidet *et al.* 2012a).

In the second study, we investigated the role of Charadriiformes (e.g. shorebirds, waders) in maintaining hotspots of AIV circulation in Afro-tropical ecosystems. Globally and locally the prevalence of AIV infection in various shorebird species sampled worldwide is classically low (<2%; (Olsen et al. 2006; Munster et al. 2007; Hanson et al. 2008; Krauss & Webster 2010) as compared with prevalence in ducks (c. 10 % globally; (Olsen et al. 2006). There is however one notable exception: a high AIV prevalence (>10%; (Kawaoka et al. 1988; Hanson et al. 2008; Stallknecht et al. 2012) has been consistently reported in the ruddy

turnstone (*Arenaria interpres*) sampled in May during spring migration at the Delaware Bay, USA. This particular species, season and site combination represent the only known shorebirds-AIV hotspot worldwide where and when infection rate is consistently higher than elsewhere in the world (Hanson et al. 2008; Krauss & Webster 2010; Stallknecht et al. 2012). Surprisingly all other shorebirds species commingling with the ruddy turnstones at the Delaware Bay in May show consistently a very low AIV prevalence (<2%;(Hanson et al. 2008; Maxted et al. 2012).

Shorebirds form the most abundant and the most species-rich group of waterbirds, but most species shared several ecological characteristics favourable to the transmission and the dispersion of AIV: i) they are generally highly gregarious (at least outside the breeding season), congregating at very high density at key staging sites along migratory flyways where they form inter-species foraging or roosting flocks; ii) most species are very long-distance migrants, including some non-stop flights of up to 11,000 km (Gill et al. 2009); and iii) many shorebirds breeding in the Northern hemisphere winter in the Southern hemisphere hence connect via migration the regions south of the equator that are not reached by migratory ducks of the Northern hemisphere.

Figure 21: Location of the study sites



(A) The Banc d'Arguin (Mauritania) and the main shorebirds migratory flyways across Western Eurasia and Africa. (B) All shorebird sampling sites considered in our study (list of sites ranked by latitude: Ukraine - Eastern Sivash, Romania- anube Delta, Turkey - Kizilirmak Delta and Yumurtalik Lagoons, Iran - Fereydoon Kenar marshes, Morocco - Marais du Bas Loukkos and Sidi Moussa-Oualidia Lagoon, Tunisia - Thyna salt pans, Egypt - Nile River Delta and Lake Qarun, Mauritania - Banc d'Arguin National Park, Senegal/Mauritania - Senegal River Delta, Republic of Sudan - El Saggay Island, Mali - Inner Niger Delta, Niger - Kurfunkura pond and Gaya, Chad - Lake Chad, Nigeria - Hadejia-Nguru wetlands, Burkina Faso - Lake Kompienga, Ethiopia - Lake Debre Zeit, South Sudan - Bargel wetland, Kenya - Lakes around Nairobi, Tanzania - Lake Manyara, Malawi - Lake Chilwa, Zambia - Kafue Flats, Zimbabwe - Lakes Manyame-Chivero, Botswana - Lake Ngami, Mozambique - Massingir Dam and Lake Chuali, South Africa - Barberspan wetland and Strandfontein). (map by M. Gély -Cirad)(Gaidet *et al.* 2012b).

We therefore specifically tested the possibility that other AIV hotspots may be associated with large shorebird congregation sites or with alternative species (Gaidet et al. 2012b). First, we conducted a survey of AIV infection in shorebirds, including ruddy turnstone, at the Banc d'Arguin in Mauritania. This site constitutes one of the largest wintering sites for shorebirds in the world (c. 2.3 million birds) and supports the greatest number of ruddy turnstones (c. 9,000 birds) across the old world (Delany, Scott & Dodman 2009). Second, we extended our analysis to a large-scale dataset of AIV prevalence in 69 shorebird species that we sampled in 25 countries at some of the most important shorebirds sites in Africa and Western Eurasia (Figure 21) during various international surveillance programs (Gaidet et al. 2007a; Cumming et al. 2011; Gaidet et al. 2012a).

Despite an unprecedentedly large geographic and taxonomic coverage we did not detect any hotspots of AIV infection in shorebirds that matched the criterion that infection rate should be consistently elevated and about an order of magnitude greater than in other sites (>10%). We did find a relatively high infection rate at one of our sampling sites - Lakes Manyame-Chivero, Zimbabwe - on three distinct occasions, representing different seasons. Follow-up sampling studies conducted at Lakes Manyame-Chivero in following years during the same season and on the same species consistently detected AIV-positive birds but at a lower infection rate, making us unable to confirm the existence of a recurrent AIV hotspot at this site. Peaks in AIV prevalence may be associated with very narrow seasonal windows e.g., few weeks in May at the Delaware Bay (Krauss & Webster 2010; Maxted et al. 2012). However the timing of these seasonal windows may be more variable in tropical than in temperate ecosystems. The high variability of seasonal rainfall in the tropics and the related fluctuations in the timing of reproduction and congregation of waterbirds may produce different seasonal dynamics of AIV infection between years. The inter-annual difference in prevalence measured at Lakes Manyame-Chivero may result from a difference in lake level and the related difference in the local density of waterbirds (Caron et al. 2011).

In summary, our study reveals that, when considered separately, the individual features associated with a disease hotspot do not systematically produce a locally and temporally high transmission rate in other contexts. Outside Delaware Bay, the ruddy turnstone has not been found infected at a higher prevalence than other shorebird species. In addition, no AIV-hotspot has been found at any of the other world's largest shorebird congregation sites investigated so far. Different constituents should be combined to generate an exceptionally high transmission rate. To what extent the constituents (species, environment, and season) of AIV hotspots are identical and temporally stable, hence predictable, remains to be elucidated. More generally, we suggest that interpreting existing hotspots in light of data from other ecosystems and pathogens should help to understand and work towards a more general model of hotspots.

Our work has therefore contributed drastically to our understanding of the drivers of AIV ecology in wild birds in Africa where before little information existed. Using the existing knowledge only available in the Northern ecosystems, we tested the main hypotheses concerning what could influence AIV prevalence in wild birds species. In addition, our work contributed as well to the general understanding of AIV in wild bird worldwide bringing new results tested on large datasets.

4. Bridge hosts for LPAI at wild/domestic bird interface

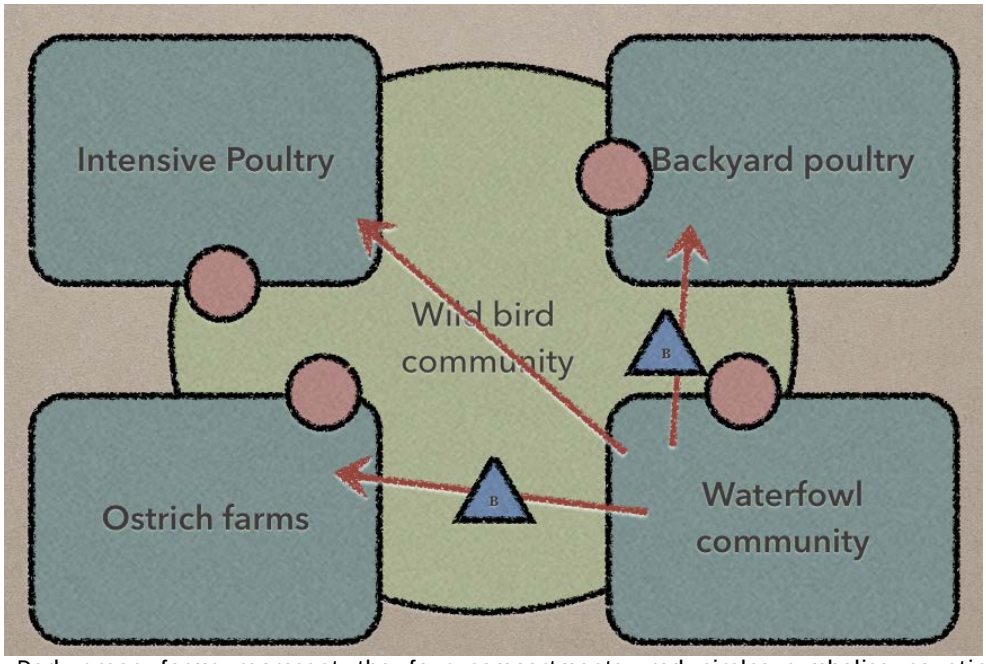
Coming back to a more applied research side of my work, all this new understanding about AIV ecology in wild birds becomes crucial when used in the light of the potential of AIV spillover to target domestic populations. In the Zimbabwean study site, I implemented longitudinal studies to formalise and implement an approach to estimate the risk of AIV transmission from wild bird to domestic populations (i.e. backyard and intensive raised poultry, ostrich farms) resulting in the definition of the bridge host EFG (see C.3) and the demonstration of the presence of bridge hosts in this SES (Caron et al. 2010; Caron et al. 2014; Caron et al. 2015a).

In the first part of this study (Caron et al. 2010), I presented a novel approach to assessing transmission risks in a complex epidemiological network that consists of spatiotemporally variable bird communities (i.e., waterbirds, domestic birds, and bridge host that interact with both wild and domestic compartments) identified as compartments regrouping sets of avian populations under similar environmental drivers (Caron et al. 2009) (Figure 22). Rather than attempting to develop a formal network-based model, we use data on the frequency and intensity of inter- and intraspecific co-occurrences, together with information about relevant aspects of species ecology and behaviour, to obtain a risk score for each species in the community and to build an adapted risk assessment model. In addition to presenting a useful picture of seasonal variation in AI risk, our analysis demonstrates how dynamic aspects of risk can still be included into epidemiological risk assessment in the absence of detailed pair-by-pair interaction data. We defined three variables: the introduction risk (IR) of AIV in the system by waterfowl; the maintenance risk (MR) of AIV in the system by waterfowl, both based on ecological traits and abundance data of waterfowl in the SES. The interaction risk (DR) represents the risk of contact between the waterfowl compartment and the three domestic compartments through wild bird using the Lakes and the surroundings of domestic compartments.

Our results provide a clear illustration of the ways in which community-level risk varies over time, both within and between years. IR peaked during the early hot-dry season, when regional waterbirds were concentrating on larger water bodies and migrants began to arrive from Europe. By contrast, MR (Figure 23) peaked in November at the end of the dry season when the largest waterbird concentrations were observed. A number of potential bridge host were shared between different epidemiological compartments, suggesting a strong potential for interactions between domestic and wild birds in this system (Figure 24).

Anseriformes and Charadriiformes represent the main families identified for IR, the former mainly as a function of their numbers and the second by their potential risk in introducing dangerous strains. Charadriiformes, mainly Palearctic waders, but also Anseriformes crossing the equator are identified by the model as potential introducers of HPAI H5N1.

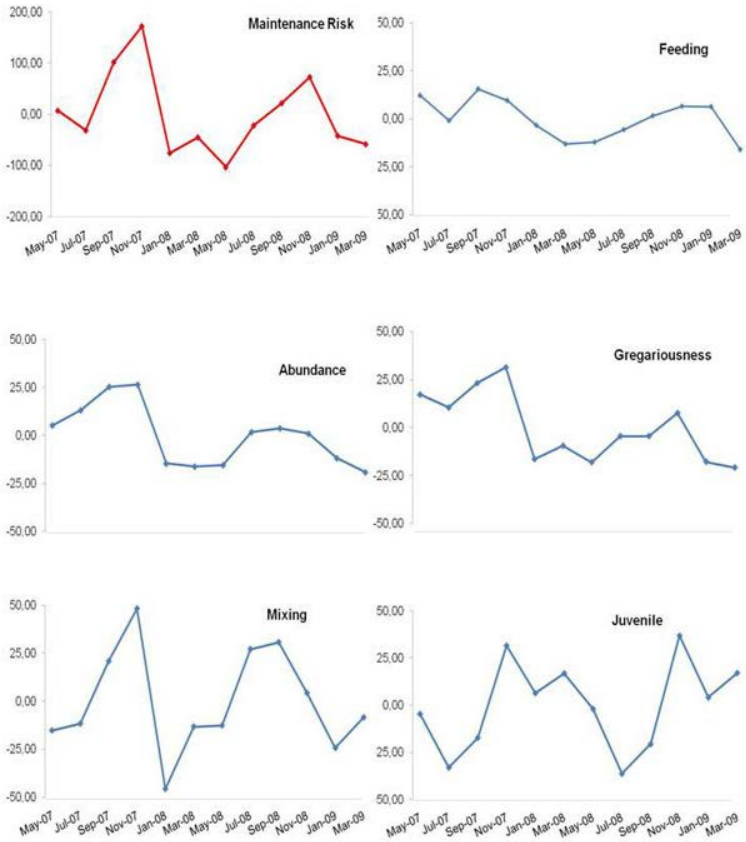
Figure 22: Model of AIV transmission between the four avian compartments in the study site



Dark green forms represent the four compartments; red circles symbolise counting session; red arrows simulate transmission pathways; blue triangle represents bridge hosts participating in some of the inter-compartment transmission events (Caron et al. 2010).

Interestingly, when waterfowl are ranked for each of the five risk factors (RFs) and the ranks are summed across the two years (Table 9), the species contributing the most to the MR belong to the bird orders known to be maintenance hosts for LPAI strains (Anseriformes and Charadriiformes) with the two most influential species in the model, the White-faced Duck and the Red-billed Teal, being the most abundant ducks in the system. The only other orders present in the 20 most important species were Gruiformes (Coot sp.) and Ciconiiformes (Egret and Ibis spp.). These orders and families have been found with, or dead of, LPAI or HPAI strains (Gauthier-Clerc, Lebarbenchon & Thomas 2007; Hars et al. 2008; Stoops et al. 2009). Additionally, the MR curve (Figure 22) was consistent across the two years and indicated a maximum risk of AIV presence in the waterfowl community during the hot-dry season, when migratory and Palearctic waterfowl are present in the system, coming from areas where AIV strains circulate.

Figure 23: Evolution of the maintenance risk (MR) and of each risk factors (RFs) that constitute it for the waterfowl compartment



Each risk factor is base on life history traits of species counted in the study site and defined as relevant for AIV maintenance in wild birds (Caron et al. 2010).

IR was not related to any peak of the DR. However, according to our model, there are always interactions between the waterfowl and domestic compartments. In a specific epidemiological situation (e.g. regional spread of a HP strains threatening the ecosystem), this IR could help to target surveillance and control measures during high interaction seasons. The fact that the highest DR curve for two domestic compartments coincided with the highest waterfowl MR is of interest (Figure 24). The end of the hot-dry season is a high risk season for these two domestic compartments, not only because the waterfowl community has the highest risk of harbouring AIV strains but also because the epidemiological interactions between the compartments are at their highest.

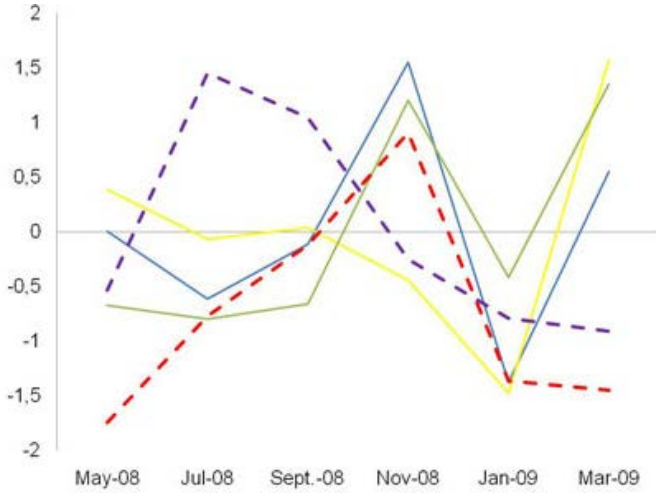
Table 9: Twenty most important species influencing the maintenance risk

Species	Family	Order	A	G	M	J	F	Maintenance Risk
White-faced Duck	Dendrocygnidae	Anseriformes	2	3	4	1	1	11
Red-billed Teal	Anatidae	Anseriformes	1	1	9	5	1	17
African Jacana	Jacanidae	Charadriiformes	5	18	1	15	1	40
Reed Cormorant	Phalacrocoracidae	Ciconiiformes	3	7	2	6	28	46
White-breasted Cormorant	Phalacrocoracidae	Ciconiiformes	7	11	7	20	28	73
Grey-headed Gull	Laridae	Charadriiformes	4	9	5	57	1	76
Black Crake	Rallidae	Gruiformes	23	42	6	21	1	93
Red-knobbed Coot	Rallidae	Gruiformes	11	5	35	26	21	98
Egyptian Goose	Anatidae	Anseriformes	13	23	20	44	1	101
Grey Heron	Ardeidae	Ciconiiformes	17	53	3	27	1	101
Cattle Egret	Ardeidae	Ciconiiformes	9	10	13	51	21	104
Glossy Ibis	Threskiornithidae	Ciconiiformes	18	27	33	7	21	106
Black Heron	Ardeidae	Ciconiiformes	19	32	24	34	1	110
Kittlitzs Plover	Charadriidae	Charadriiformes	8	8	41	41	27	125
Common Moorhen	Rallidae	Gruiformes	31	45	18	36	1	131
Spur-winged Goose	Anatidae	Anseriformes	43	17	61	2	17	140
Yellow-billed Egret	Ardeidae	Ciconiiformes	32	63	17	30	1	143
African Sacred Ibis	Threskiornithidae	Ciconiiformes	21	25	36	33	38	153
Squacco Heron	Ardeidae	Ciconiiformes	27	62	8	56	1	154
Southern Pochard	Anatidae	Anseriformes	36	28	53	11	41	169

Ranked per risk factors (decreasing ranking) and sum across the 5 RFs values for the last column; A = Abundance dynamic RF; G = intra-species mixing dynamic RF; M = inter-species mixing dynamic RF; J = proportion of juvenile in the population dynamic RF; F = feeding non-dynamic RF (Caron et al. 2010).

The second peak after the end of the rainy season (in March) was consistent for the three domestic compartments but was not linked with a peak in risk associated with the waterfowl community. However, the shared community of wild birds between the waterfowl community and the three domestic compartments was always high suggesting a yearlong risk of pathogen transmission from the waterfowl compartment. The validity of the DR estimate is limited by its population-level approach; birds of the same species observed in two different compartments were assumed to belong to the same population. However, we cannot prove that they were indeed the same individuals beyond the fact that the study site is fairly small.

Figure 24: Variation in the interaction Risk (DR) for each domestic compartment



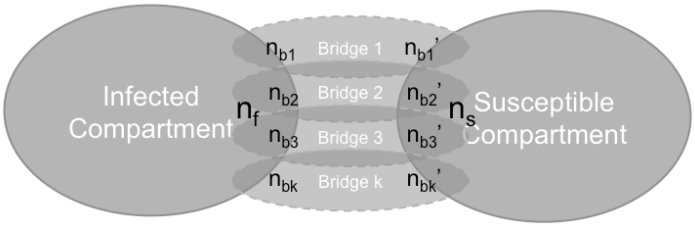
Plain lines= intensive poultry –blue, backyard poultry – yellow, ostrich farms-green) associated with introduction (IR) and maintenance risk (MR) for the waterfowl community (dashed lines, IR-purple, MR-red) (Caron et al. 2010).

The protocol described here is intensive but feasible. Its approach could easily be simplified and reproduced. In the context of AIV surveillance, a series of counts by ornithologists during suspected high-risk seasons would prepare the ground for targeted sampling. In some countries, this type of data is regularly collected by ornithological organizations and therefore already available. The strength of this research relative to traditional epidemiological analyses lies in its ecological dimensions. Although our model was designed with the ecology of AIV in mind, most pathogens with direct transmission will be dependent on the ecological traits estimated by the RFs (with some adjustments, e.g., "Feeding" RF). Can this risk factor analysis be extended to other pathogens to develop more 'ecological' predictions of disease risk? Such approaches may ultimately provide useful guidelines for surveillance in hotspots of disease emergence at the wildlife/domestic interface (Jones et al. 2008).

The second part of the study (Caron et al. 2014) was to identify bridge hosts in this ecosystem based on the bird count dataset implemented during 2 years at domestic and wildlife compartments (same datatset as previously). This protocol followed the line of thoughts presented in C.3 (albeit the final article was published after) defining a framework to identify bridge hosts in multi-host systems (Caron et al. 2015a). In the first part of the study, the EFG framework was not applied to bird species observed. Therefore maintenance hosts (Anseriformes and Charadriiformes) were included in the risk factor analysis and because of the knowledge on their role in AIV ecology and their abundance in the ecosystem, the model identified them as most at-risk species for transmitting AIV to poultry. However, in the second study, we distinguish between maintenance and non-maintenance hosts, therefore being able to focus on species' populations able to bridge maintenance and target populations. In addition to the 3 domestic compartments already presented, the waterfowl compartment regroups Anseriformes and Charadriiformes and the rest of the wild bird community gathers a pool of populations potentially connecting the 4 compartments and bridging the epidemiological gap between them if existing (Figure 22).

I focused the study on the risk of AIV spread by direct or indirect contact between wild and domestic birds, although I acknowledge the fact that other transmission pathways could be eligible (e.g. poultry trade, human vector). First I used bird counts to identify wild bird species potentially playing a bridge role in the ecosystem, allowing me to quantify the relative proportion of potential contacts between maintenance, bridge and target hosts and reducing the multi-host complexity by ranking species the most at risk of playing a bridge role. I calculated IS, the Interaction Sum, a proxy of inter-compartment contacts through wild birds (Figure 25). Then, I conducted targeted sampling on the species identified to investigate their exposure to AIV when present in the ecosystem during high-contact season. Therefore, instead of sampling "blindly" within the wild bird community, this prioritization process can help guiding AIV surveillance efforts towards the most likely bridge hosts and during seasons at risk.

Figure 25: Scheme representing how the Interaction Sum was calculated



$$IS_{bridge} = \sum_{(i:1 \rightarrow k)} (n_{bi} * n_{bi}')$$

Schematic representation of the role of multiple bridge candidates between an infected and a susceptible avian compartment and showing how the Interaction Sum (*IS*) variable was calculated. *ni* = the number of birds in the infected compartment; *ns* = the number of birds in the susceptible compartment; nb_1 = the number of birds in bridge species population 1 in contact with the infected compartment; nb_1' = the number of birds in bridge species population 1 in contact with the susceptible compartment (Caron *et al.* 2014).

Our two and half-year study demonstrated that: 1) the shared community of bridge candidates varies significantly according to the interfaces or pairs of bird compartments considered; 2) no seasonal nor inter-annual variability trends of the variable IS have been detected in this shared community; 3) potential indirect contacts between maintenance and target species through bridge hosts are 20-fold more frequent than potential direct contacts between maintenance and target populations; 4) a few dominant bridge candidates represent most of the risk of contact between pairs of compartments despite hundreds of species observed; 5) Two out of three of the most co-occurring bridge candidates did harbour AIV genetic material in the agro-ecosystem studied, proving their bridge role in this ecosystem.

Bridge candidates were not distributed equally between compartments. Ecological, environmental and anthropological factors can influence the distribution of these species. The distance between compartments could also be a confounding factor as explained in the previous study. Variability between sites belonging to the same compartment has not been taken into account in the analyses as it was assumed that sites of the same compartment (i.e. same production or natural systems) shared characteristics such as availability of

resources or roosting potential. Domestic production systems provide attractors for wild birds. Ostrich are fed and watered outdoor in large drums that wild birds can easily access. Intensive poultry are fed indoor but food leftover outside buildings or in open buildings during quarantine periods can provide food resources to wild birds. On the contrary, backyard poultry are left foraging within and outside villages and compete with wild birds for "natural food resources". Buildings can also provide roosting site for passerine species such as swallow species as was often observed during this study. Therefore, we believe that compartment-specific characteristics tend to define the community of wild bird species using these compartments.

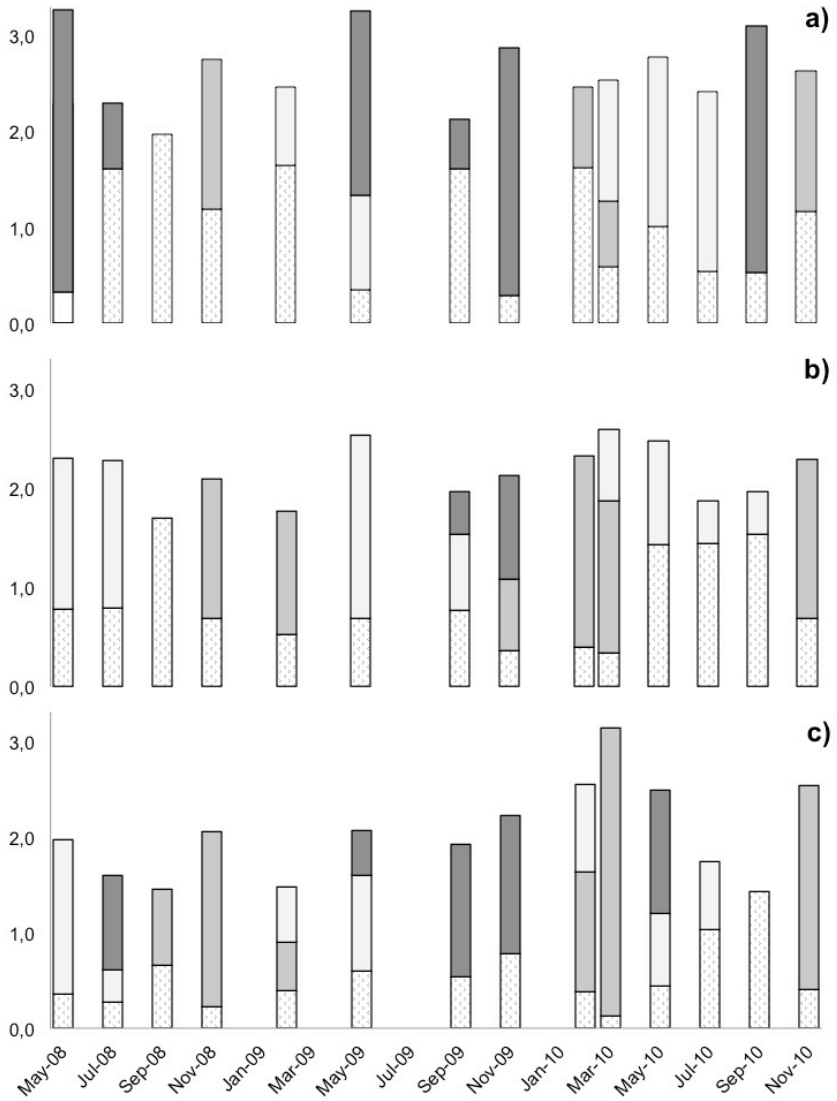
No significant difference of IS (Interaction Sum) or species richness was observed between seasons or between years. This result is particularly counter intuitive as wild bird experience seasonal population dynamics due to movements (e.g. migration) or reproduction (Mundava et al. 2012). As mentioned already, rainfall patterns in sub-tropical Africa are more variable in time and space than in the northern hemisphere and wild birds adapt their behaviour to this variability through complex behaviour such as nomadism (Verschuren, Laird & Cumming 2000; Dodman & Diagana 2007). For example, red-billed queleas (*Quelea quelea*) are nomadic birds responding to resource availability determined by rainfall and the timing of their arrival in a specific ecosystem can vary (e.g. May 2008 & 2009 but not in May 2010 in Figure 26)(Dallimer & Jones 2002). Palaearctic migrants such as barn swallow (*Hirundo rustica*) leave Eurasia to arrive in Zimbabwe in September to depart again around March and April. If no seasonal or inter-annual trends were observed at the IS level, at the (dominant) species level, wild bird ecology induces periodic trends in potential contacts. The low variability of species richness across season and years supports the hypothesis that domestic compartment "artificially" attract wild bird species, as the species richness varied on the lake shores in a previous study (Caron et al. 2010).

We measured the potential direct contacts between the maintenance and target domestic hosts: they represented between 4.0 and 5.1% during the intensive protocol (see details of both intensive and longitudinal protocols in the article) of the potential indirect contacts through bridge candidates. Some of the domestic compartments we have monitored during this study were close to or on the lakeshores: direct contacts between maintenance and target species were more likely to occur under these circumstances. Maintenance hosts should experience a higher prevalence and a better propensity to transmit AIV to target populations and could therefore trigger less but more efficient infectious contacts.

Only 12 bridge candidate species (9 Passeriformes species, two Ciconiiformes and one Columbiforme) were identified as dominant (i.e. representing more than 20% of IS during at least one session) during the intensive and longitudinal protocol in which 165 and 230 bird species were observed including 15 and 33 maintenance species respectively. Firstly, it indicates that the majority of potential contacts between compartments could be managed by concentrating efforts on a limited number of candidate bridge hosts. Of interest is the presence of 6 swallow-type species, which are probably attracted to farming systems by the same resources (e.g. insects, roosting) and may therefore be managed by the same measures. Interestingly, proofs of AIV susceptibility for the three species dominating the potential contacts between compartments exist in the literature (Gronesova et al.

2008b; Mizakova, Gronesova & Betakova 2008; Breithaupt et al. 2010; Phuong et al. 2011). Barn swallow was also identified as potential bridge host in a very different ecosystem (Burns et al. 2012). Our PCR results confirmed the role of bridge host in our agro-ecosystem for barn swallow and quelea, but not for cattle (Bulbucus ibis). These results seem to support that the proposed framework based on count data is an efficient approach to identify potential bridge host. However, these results could result from the fact that most wild bird species are competent for AIV. In which case, the framework presented here would still be valid, as it identifies the hosts that have the highest intensity/frequency of contacts with maintenance and target population and therefore highlights high-risk bridge hosts for AIV transmission.

Figure 26: Variation of the log of IS in the longitudinal protocol

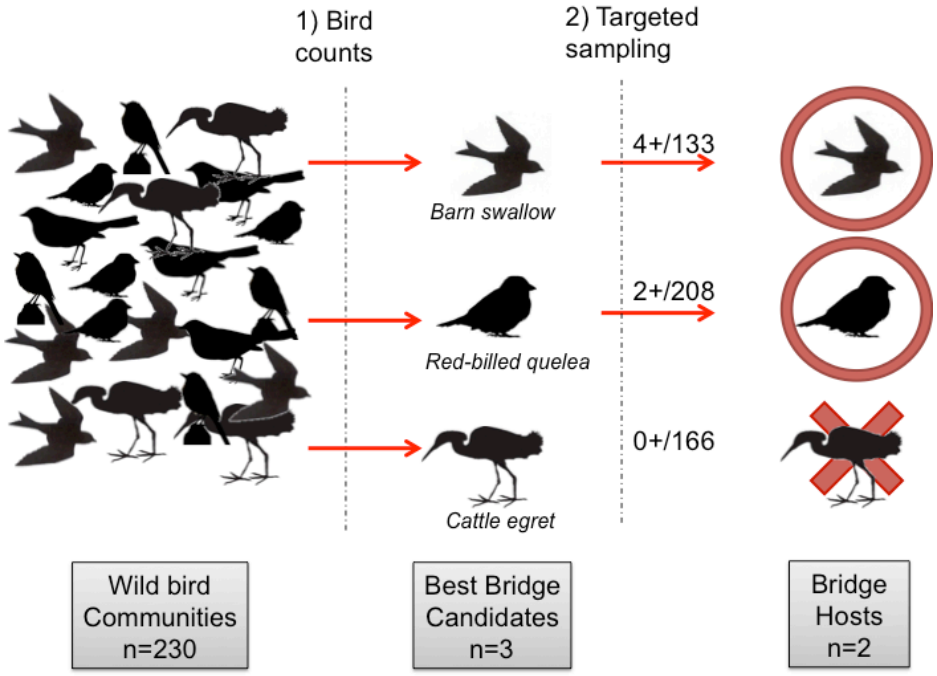


The proportion of the three main dominant species is indicated: in dark grey, red-billed quelea; in medium grey, barn swallow and in light grey cattle egret. Dash bars indicate the rest of the bird community. In (a) the backyard/intensive poultry interface; in (b) the backyard poultry / waterfowl interface and in (c) the intensive poultry / waterfowl interface (Caron *et al.* 2014).

Our results have direct implication for the management of the AIV risk in the agro-ecosystem. For example, red-billed queleas are considered as a crop pest in

southern Africa and a variety of control options are currently used to avoid those visiting crops, which could also be used to limit contacts with domestic poultry. Barn swallows are probably mainly visiting production units to feed on insects. Insect control options may for example reduce these visits. Modification of the habitat could also reduce roosting sites at proximity of production buildings. Cattle egrets are often following cattle in the proximity of farms. Avoiding cattle visiting to those farms (as it has been often observed during field visits) would reduce the interface. A few control options could therefore significantly reduce the risk of spread of AIV between compartments. However the inter-compartments variability would require adapted management depending on production systems.

Figure 27; Schematic representation of the eco-epidemiological approach



In order to reduce the complex multi-host system of the wild bird community, bird counts identify the bridge candidates with the highest risk of bridging the two compartments studied. Here 3 bird species are identified as such and targeted and adapted sampling and AIV testing on these species confirm (barn swallow & red-billed quelea) or not (cattle egret) their bridge role in the agro-ecosystem (Caron *et al.* 2014).

This eco-epidemiological framework should be used to investigate the neglected role of bridge host in AIV epidemiology, a role that could be determinant in the local spread of the disease between avian populations and even towards humans (Jones et al. 2008). The bird count protocol presented here is time consuming (i.e. 584 hours of bird counts) but requires only ornithological skills, which may be obtained at low/no cost in many areas through volunteer participation of ornithological associations. In addition, available local ornithological datasets could be used to prioritise bridge candidate amongst the avian diversity. Instead of sampling blindly the avian community, this framework can structure surveillance by targeting the bird species with the highest contact rates between pairs of compartments as these species are the most likely to spread diseases. From a theoretical point of view, it would be interesting to investigate the shared

communities of bridge hosts in other ecosystems to assess if our findings (i.e. that most of the potential contacts are made by a few species) are site specific or not.

Finally, as this approach is based primarily on potential contact and co-occurrence pattern, it could be used for other pathogens and different animal models, by simply adapting the definition of "maintenance" and "target" populations to the pathogen considered (see section F.1). This is an important advantage of this method and the methods based on functional approaches as we present them.

5. Challenging the AIV maintenance dogma in wild birds

The experience that we accumulated from working on AIV ecology in wild birds and at wildlife/domestic interface in new ecosystems (*vs.* Northern ecosystem) in Afro-tropical regions put us in a position where we can now re-assess the global paradigm in this thematic. We suggest that the geographically and taxonomically biased sample size of AIV data in wild bird worldwide has led to paradigms adapted to these ecosystems but probably not suited for elsewhere. This observation is of course of importance for the implementation of AIV surveillance in wild birds and at wild/domestic interfaces.

"Anseriformes and Charadriiformes are the major natural reservoir/maintenance hosts for Avian Influenza viruses in natural ecosystems". This sentence commonly informs introductive sections of articles addressing issues related to AIV and wild birds since the publication of major reviews ten years ago (Olsen et al. 2006; Fouchier et al. 2007). The role of maintenance for a host population is difficult to prove in vivo, however this concept is often linked in AIV epidemiology with species harbouring high and recurrent prevalence of AIV with low observed mortality.

Wild ducks, especially species of the Anas genus foraging by dabbling, have been consistently reported with the highest AIV prevalence in various ecosystems and regions of the world (Munster et al. 2007). The AIV prevalence in Charadriiformes is however usually lower (c. 1%; (Olsen et al. 2006; Krauss & Webster 2010; Gaidet et al. 2012b), at a level similar to other birds groups much rarely tested. Several elements have led to the understanding that Charadriiformes are important players in AIV ecology. First, the first detection of AIV in wild birds was in a Charadriiformes species, the Common terns (Sterna hirundo), during an outbreak in 1961 in South Africa (Becker 1966). This preliminary finding was then consolidated by work on shorebirds and gulls at the Delaware Bay in the north-eastern USA where these birds were found to harbour specific AIV subtypes not found in wild ducks (Kawaoka et al. 1988). The high isolation rate of AIV consistently found in ruddy turnstone during spring migration (already presented in the previous paragraphs) since earlier studies fuelled the interest for investigating AIV in Charadriiformes (Krauss et al. 2010). Continuous annual surveillance for AIV at the Delaware Bay since 1985 provided the bulk of our understanding of AIV in Charadriiformes. The presence of larger variety of hemagglutinin and neuraminidase combination subtypes in Charadriiformes than in Anseriformes, including hemagglutinin subtype detected exclusively in gulls (Fouchier et al. 2005), has been confirmed by subsequent studies (Krauss et al. 2004). Gulls have also shown some seasonal peaks in prevalence (Toennessen et al. 2011; Verhagen

et al. 2014). These pioneer research findings logically focused the attention of research and manager communities on Charadriiformes.

Since 2005, many studies across the world have targeted major wild bird communities for AIV sampling. Avian diversity in most ecosystems can reach several hundreds species and the global avian diversity is around 10 000 species in 249 families and 46 orders (Boyd 2015). In order for surveillance protocols to be epidemiologically sound (e.g. 299 samples of the same species to have a 0.95 probability of detecting a 1% prevalence) and still fit within budget and time constraints, a prioritization process was necessary and often resulted in the targeting of Anseriformes and Charadriiformes, based on available knowledge. Catching wild birds requires implementing trapping or netting techniques often catching non-target species. Most of the time, the sample size for these by-catch species are small and the epidemiological insight given by these results is weak. Only a positive sample for AIV provides an indication on the exposure and susceptibility of the species to AIV infection.

However, the number of studies focusing on the role of Anseriformes and/or Charadriiformes implemented recently across the world has produced a large amount of “by-catch” information about non-Anseriformes and non-Charadriiformes species. In addition, a growing number of studies reporting on the detection of AIV in bird species non-conventionally recognised has maintenance hosts (e.g. passerines) raises the question of the roles of other bird groups in AIV ecology (Peterson et al. 2008; Caron et al. 2012b; Thinh et al. 2012; Williams et al. 2012; Zhao et al. 2014).

We screened the scientific literature to gather and aggregate all the data available on AIV infection rates in wild birds tested by PCR. Information on AIV infection rate in various bird groups had been previously exhaustively collected in two earlier reviews published by Alexander (Alexander 2000) and Olsen et al. (Olsen et al. 2006). We used detailed information on non-Anseriformes and non-Charadriiformes PCR results for AIV from these two reviews and complete the database by searching all available scientific literature published from 2006 to 2012. Using Google Scholar in order to access a wide range of published data, keywords associated with "influenza" were used to search available literature (e.g. "waterfowl", "wild bird", "terrestrial bird"). For each search more than 200 records were screened by title (after 100 records, the majority of the articles were irrelevant) and selected if they could provide information on non-Anseriformes and non-Charadriiformes PCR testing for AIV. When data on non-Anseriformes and non-Charadriiformes PCR testing for AIV was found, the following information was collected: name of first author; year of publication; year of data collection; source of information (i.e. literature. report); site (country); region (by continent); bird species (and latin name), family, order; number of samples; number of PCR positive samples; sample type (tracheal, cloacal, both or collected on the ground from identified species). When available, type of pathogenicity and strain information was also collected.

In order for the data to be comparable and not to induce bias in the database, we decided to take into account only data from healthy wild birds. Birds found dead or showing symptoms were excluded from the database. Data related to outbreak investigations (especially HPAI H5N1) was also discarded. This decision was taken on the basis of the distinct ecology of highly virulent AIV in wild birds,

mostly resulting from the spillback of HPAI form poultry to wild birds and for which maintenance by wild birds is not suspected (Feare 2007). When information was not clear, focal persons were contacted to clarify the data type, source and collection.

The database contains results for 69,566 non-Anseriformes and non-Charadriiformes birds tested for AIV by PCR, including 1,056 bird species, 121 families and 25 orders in 98 scientific sources (mostly scientific publications and new data from our research unit unpublished). Out of this sample, 815 birds tested positive for AIV by RT-PCR, indicating an infection rate of 1.17% (95% confidence interval: 1.09% - 1.25%). Table 10 presents the number of birds found positive, the sample size, infection ratio and its confidence interval for all the 25 bird orders investigated. A total of 15 out of 25 bird orders had a samples size large enough to detect at least one positive bird if the global infection rate in this bird group is >1%. The AIV infection rate in eight of these bird orders is higher than the one reported globally for Charadriiformes (Olsen et al. 2006). Out of the 25 bird orders represented in our dataset, 18 have at least one positive sample for AIV and out of the 7 left, only one has a sample size that could detect a prevalence of 1% (i.e. Sphenisciformes. n=1133),. Amongst the 45 species having a sample size superior to 300, 32 (71%) had at least one positive sample for AIV and 12 (27%) had an infection ratio close or above 1%, close to the Charadriiformes' infection ratio in Olsen et al. (2006).Prior to 2006 (the reference point being the publication of Olsen et al. 2006 which coincides fairly well with the upsurge of interest on AIV in wild birds), 37,491 non-Anseriformes and non-Charadriiformes wild bird samples had been collected for an infection rate of 1.10%. For the period of 2006 - 2012, these figures are 32,075 and 1.26%. In Figure 28, the relationship between sample size and infection ratio is displayed by bird order and species, including values given by Olsen et al. (Olsen et al. 2006) for Anseriformes and Charadriiformes.

Table 10: Number of AIV positive by RT-PCR and sample size (n) by bird orders

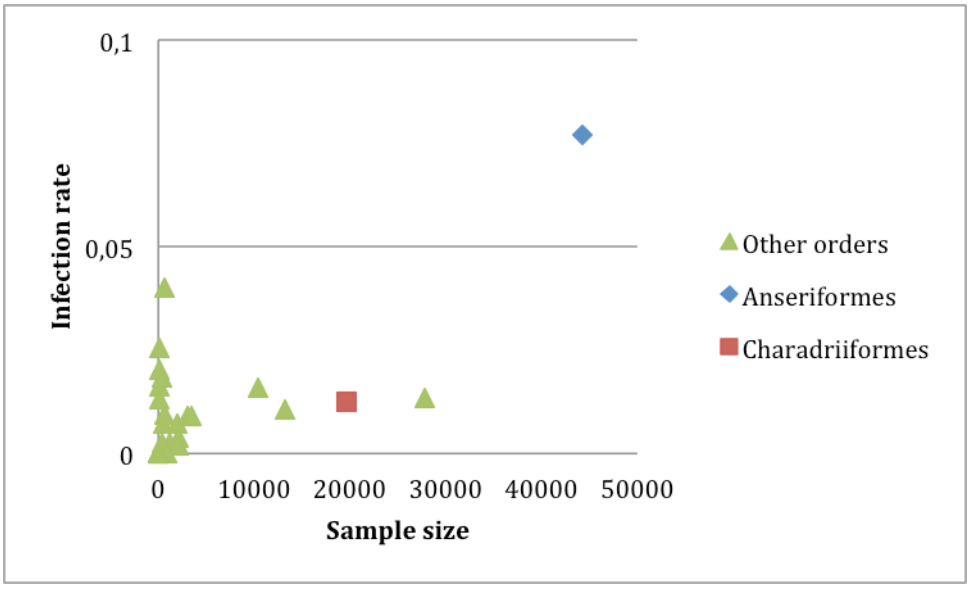
Order	IAV positif	n	% of IAV +	Confidence interval (95%)
Anseriformes (ducks, geese)	3416	44318	7.71	7.46-7.96
Phoenicopteriformes (flamingos)	26	648	4.01	2.50-5.52
Apodiformes (swifts)	3	118	2.54	0.00-5.39
Gaviiformes (loons)	1	49	2.04	0.00-6.04
Podicipediformes (grebes)	8	436	1.83	0.57-3.10
Cuculiformes (cuckoos)	1	62	1.61	0.00-4.77
Gruiformes (cranes, coots)	164	10396	1.58	1.34-1.82
Passeriformes (passerines)	370	27838	1.33	1.19-1.46
Psittaciformes (parrots)	1	77	1.30	0.00-3.84
Charadriiformes (waders, gulls)	244	19663	1.24	1.09-1.40
Pelecaniformes (pelicans)	143	13285	1.08	0.90-1.25
Strigiformes (owls)	6	629	0.95	0.19-1.71
Galliformes (quails, grouses)	31	3439	0.90	0.59-1.22
Columbiformes (doves, pigeons)	27	3001	0.90	0.56-1.24
Coraciiformes (kingfishers, bee-eaters)	4	558	0.72	0.02-1.42
Accipitriformes (raptors)	14	1968	0.71	0.34-1.08
Ciconiiformes (egrets, storks)	8	2154	0.37	0.11-0.63
Falconiformes (falcons)	3	1182	0.25	0.00-0.54
Piciformes (woodpeckers, barbets)	1	396	0.25	0.00-0.75
Procellariiformes (shearwaters,	4	2148	0.19	0.000.37

petrels)				
Caprimulgiformes (nightjars)	0	17	0.00	0.00-0.00
Coliiformes (mousebirds)	0	33	0.00	0.00-0.00
Otidiformes (bustards)	0	65	0.00	0.00-0.00
Pteroclidiformes (sandgrouse)	0	64	0.00	0.00-0.00
Sphenisciformes (penguins)	0	907	0.00	0.00-0.00
Tinamiformes (tinamou)	0	84	0.00	0.00-0.00
Trogoniformes (trogon)	0	12	0.00	0.00-0.00
Total *	**815**	**69566**	**1.17**	**1.09-1.25**

Ratio represent the ratio of the 2 previous variables (expressed here in %) and "min" and "max" represent lower and upper values for the 95% confidence interval. Grey lines indicates results for Anseriformes and Charadriiformes extracted from Olsen et al. (2006) for reference (Caron, Cappelle & Gaidet-Drapier Submitted).

Contrary to the current dogma, our results suggest that a wider diversity of bird groups may play a particular role globally for AIV epidemiology. We found that several bird orders had globally a higher AIV infection rate than Charadriiformes, so far considered as the second most important host group for AIV after Anseriformes. The recognition of Charadriiformes as maintenance host for AIV has been based on: (i) literature review indicating a global infection rate of 1.24% (Olsen et al. 2006); (ii) the site-specific studies at the Delaware Bay where prevalence in Charadriiformes has been consistently high (Krauss et al. 2010); (iii) the isolation of a wide diversity of AIV in Charadriiformes including subtypes detected exclusively in gulls (Fouchier et al. 2005). Our database demonstrates that the first point (i) can be challenged, as there is a range of bird orders that display similar global infection ratio (4 orders with a sample size above 300). At the species level, some common bird species also show a relatively globally high infection rate. For instance the barn swallow, tested in 16 different studies sampled on 3 continents (North America, Europe and Africa), was found with an infection rate of 4.28% (n=327). The Eurasian magpie (*Pica pica*) has been sampled on 2 continents in 8 studies shows a global infection rate of 13.23% (n=136). The eurasian coot (*Fulica atra*) has been sampled in 46 different studies for an infection rate of 2.07% (n=5641). One can notice that some AIV have been isolated in each of these three bird species (Gronesova et al. 2008a; Gronesova et al. 2008b; Gronesova et al. 2008c; Fereidouni et al. 2010; Marchenko et al. 2012). For comparison with Olsen et al. (Olsen et al. 2006), the highest infection ratio at the species level in Charadriiformes was 2.2% in the Black-headed Gull (*Larus ribundus*, n=770) (Figure 28).

Figure 28: Infection rate of AIV detected by RT-PCR in relation to the number of sample tested for each bird order



Anseriformes and Charadriiformes infection rate values from Olsen et al. 2006 are presented in blue and red respectively (Caron, Cappelle & Gaidet-Drapier Submitted).

For most non-Anseriformes and non-Charadriiformes orders, no sample size large enough have been tested at a population level to inform points (ii) and (iii) on the potential for some bird groups or species to act as maintenance host for AIV. In addition, because protocols were targeting primarily Anseriformes or Charadriiformes, non-Anseriformes and non-Charadriiformes species may not have been sampled at a time when their population have the highest prevalence, limiting our understanding of their role in AIV epidemiology. In Charadriiformes, one can notice that no other site-specific hotspot of AIV infection (point (ii)) have been detected so far outside the Delaware Bay (Krauss & Webster 2010; Gaidet et al. 2012b).

The interpretation of this dataset needs to be taken carefully, understanding its bias. Firstly, regrouping samples from different populations, species and families, from different regions, sites and collected at different seasons may mask some species and spatio-temporal variability in population prevalence and some species-specific receptivity. Despite these inherent limits, this approach has been used previously to synthetize and discuss AIV rate of infection across bird families and orders (Alexander 2000; Olsen et al. 2006). Secondly the information related to data included in the dataset could be questioned. Only data from PCR analysis was taken into account but PCR testing in different laboratories can have variable sensitivity. In addition, identically, each result does not refer to the same type of sample and, in 17.5% of the dataset, the type of sample collected (i.e. tracheal, cloacal) was not specified. The species and "family and species" levels were not indicated in 15.4% and 8.5% of the dataset respectively as well. We strongly recommended that the minimum information presented here should be reported when sampling wild birds in order to provide the maximum possible information (Yasue et al. 2006). Additional 30,000+ AIV PCR results could be extracted from official reports and online database from our on-line search. However, we could not with certainty assess if these results overlapped with peer-reviewed studies

and if the context in which samples were collected meet the conditions to enter our dataset (e.g. data collected during HPAI H5N1 outbreak do not qualify). However, including this data in our analysis achieves a 100,000+ PCR result dataset comforting the results presented here.

Recent studies have investigated the circulation of AIV in other bird orders/families (Peterson et al. 2008; Thinh et al. 2012; Williams et al. 2012; Ramey et al. 2014; Slusher et al. 2014; Zhao et al. 2014) but remain too scarce. More recent testing of the hypothesis of a hotspot of AIV circulation in high-density Charadriiformes communities failed to replicate the observed patterns in Delaware bay (Krauss et al. 2010; Gaidet et al. 2012b). Nevertheless, Charadriiformes harbour specific AIV subtypes that are not detected in other bird orders, suggesting a potential role in maintaining these strains. Long-term investigations in other bird orders are necessary to conclude if this pattern is specific to Charadriiformes or not. Recently, H17 and H18 AIV were detected in bat species (Tong et al. 2013) indicating that much remains to be discovered in AIV diversity and host range.

Our main message is not to question the role of shorebirds and gulls has important hosts for AIV, but rather to call for reconsidering the role of other bird group in the transmission, maintenance and diversity of AIV. We believe that the current paradigm about Anseriformes and Charadriiformes being assumed to be the main maintenance hosts drives the attention mainly towards two bird orders and prevents the evolution of our understanding of AIV ecology in wild bird communities as a whole.

The alternative hypothesis to explain the maintenance of AIV in wild birds is based on the concept of maintenance community (Haydon et al. 2002). A maintenance community is one or more epidemiologically connected populations in which the pathogen can be permanently maintained. Given the wide range of bird species susceptible for AIV and able therefore to play a role in the maintenance of these pathogens, the specific composition of the avian community, its species diversity and abundance will determine the potential for AIV maintenance in a given ecosystem. In ecosystem where duck species occur at high relative abundance within the avian community, the maintenance of AIV is most probably driven by ducks species (Latorre-Margalef et al. 2014). At Delaware Bay, shorebirds, gulls and ducks are the most abundant avian species the avian community and drive AIV dynamics locally and most probably influence AIV dynamics in the region (Krauss et al. 2010). We argue that, following this argument, in avian communities where non-Anseriformes and non-Charadriiformes bird species represent the highest abundance, AIV can also be maintained by one or most probably a subset of species' populations interacting in this ecosystem (Caron et al. 2012b). For example, Passeriformes include highly abundant and social species such as the red-billed quelea that has been shown to be susceptible for AIV experimentally and in nature (Breithaupt et al. 2010; Caron et al. 2015a). In ecosystems where they occur, red-billed quelea colonies should be investigated for AIV prevalence. Similarly, swallow sp. are a group of species including some Palearctic migrants that can roost in millions providing in some ecosystem an ideal multi-host system where AIV can be transmitted between hosts and be maintained. More recently, isolation in a tree sparrow (*Passer montanus*) of the newly emerged H7N9 virus in China where it caused severe human disease, and the evidence of replication of this H7N9 virus in several terrestrial birds, raised concerns about the

potential role of some passerines in the transmission of AIVs with a pandemic potential (Jones et al. 2014; Zhao et al. 2014).

This alternative hypothesis calls for the revisiting of how AIV wild bird studies are currently designed. Firstly, avian communities with low abundance or absence of Anseriformes and/or Charadriiformes should also be the focus of longitudinal studies to confirm or not if they can maintain AIV. This entails investigation in new ecosystems including non-wetlands. Second, Anseriformes and Charadriiformes targeted sampling should not be done a priori, especially for the later. In focal ecosystems, information on the bird community and its seasonal dynamics should be gathered or collected. Ecological traits (e.g. abundance, social behaviour, migration patterns) and available epidemiological data (e.g. using a database like the one presented here) should be merged to identify species that can potentially play a role in the local maintenance of AIV (Cumming et al. 2008; Caron et al. 2014). Then hypotheses should be designed on the most probable maintenance hosts in the ecosystem and targeted epidemiological sampling using species-specific capture techniques should help testing these hypotheses with proper sample size per species.

If applied, this new framework should produce robust epidemiological data on non-Anseriformes and non-Charadriiformes species able to re-assess our understanding of AIV ecology in wild birds,. A better understanding of the role of wild birds in the maintenance of AIV as well as other epidemiological roles (e.g. bridge host (Caron et al. 2014; Caron et al. 2015a) will help the design of surveillance and control options to prevent the spillover of AIV to domestic poultry and ultimately humans. The maintenance and strengthening of the database on non-Anseriformes and non-Charadriiformes bird species presented here should be pursued in order to provide the basis for further research on the potential maintenance hosts for AIV.

Using a disease ecology approach as presented in section C, helped me and us at AGIRs as a team to significantly improve the knowledge on AIV in wild birds and at the wild/domestic bird interface. This improvement applies not only within our SES, but also to Africa and worldwide. In addition, this approach helped us develop tools and framework ready to be replicated in other multi-host systems and other disease models as presented briefly in a later section.

E. Wild and domestic ungulates, human and diseases in TFCAs

I have been working on this second model since 2006 but this work echoes previous works on the transmission of pathogens at wildlife/domestic interfaces in Africa (Caron, Cross & du Toit 2003; Caron & Kock 2004; Etter et al. 2006). In Transfrontier Conservation Areas (TFCAs) of southern Africa, protected areas occur in a complex matrix of land-use types, often in contact with communal lands (Binot, Castel & Caron 2006). Hence, different types of interface areas can create opportunities for pathogen inter-species spillover between hosts. These transmission events can lead to pathogen and/or disease emergence or endemism of some pathogens within the wild and domestic host community (Artois et al. 2006).

Are these diseases important locally and threatening local livelihoods and wildlife conservation? A study we implemented in communal zones close to protected areas in Zimbabwe using participative tools (i.e. free-listing method) indicated that small-scale farmers were particularly concerned by diseases of their livestock and that for the most important of these diseases they were convinced that wildlife can play a role in the transmission of these diseases to their heads of cattle (de Garine-Wichatitksy et al. 2013c)(Table 11).

Table 11: Most frequently cited livestock diseases by cattle owners in Zimbabwe

Disease/syndrome	Disease citation	Do not know	No	Yes	Species citation	Main wildlife sp. cited	Second most cited wildilfe species
Foot and mouth	190	18%	4%	77%	171	Buffalo (82%)	Wildebeest (6%)
Lumpy Skin	127	52%	26%	22%	35	Buffalo (54%)	Wildebeest (17%)
Blackleg	122	39%	29%	32%	50	Buffalo (62%)	Wildebeest (14%)
Heartwater	89	39%	37%	24%	26	Buffalo (62%)	Wildebeest (11.5%)
Anthrax	67	46%	22%	31%	34	Buffalo (47%)	Wildebeest (15%)
Babesiosis	43	33%	47%	21%	10	Buffalo (80%)	Eland, Elephant (10%)
Anaplasmosis	42	38%	33%	29%	14	Buffalo (57%)	Elephant (14%)
NID	42	48%	24%	29%	14	Buffalo (64%)	Greater kudu, Wildebeest (14%)
Rabies	38	8%	0%	92%	41	Jackal (78%)	Wild dog (12%)
NID Limping	28	46%	32%	21%	6	Buffalo (67%)	Eland, Elephant (17%)
Brucellosis	23	39%	48%	13%	4	Buffalo (50%)	Greater kudu (25%), All wild animals (25%)
Foot Rot	19	42%	37%	21%	4	Buffalo (75%)	All antelopes (25%)
Mastitis	17	53%	35%	12%	5	Buffalo (40%)	Eland, Greater Kudu Sable (20%)
NID Ocular	16	44%	44%	13%	3	Buffalo (67%)	Elephant (33%)
NID Neurological	15	40%	53%	7%	0	NA	NA
NID Cutaneous	14	50%	29%	21%	4	Bushpig (50%)	Wildebeest (50%)
NID Diarrohea	14	57%	29%	14%	2	Buffalo (100%)	NA
Ticks	13	46%	0%	54%	9	Wildebeest (33%)	Bushpig (22%)
Dermatophilosis	10	40%	20%	40%	4	Buffalo (50%)	Bushpig, Wildebeest (25%)
NID Digestive	10	50%	30%	20%	1	Buffalo (100%)	NA
NID Paralysis	10	40%	0%	60%	7	Buffalo (71%)	Impala, Warthog (14%)
Newcastle	8	63%	25%	13%	1	Buffalo (100%)	NA
Contagious Opthalmia	7	14%	57%	29%	2	Buffalo (100%)	NA
NID Salivation	7	57%	43%	0%	0	NA	NA
Theileriosis	7	57%	0%	43%	5	Buffalo (60%)	Wildebeest, Elephant (25%)

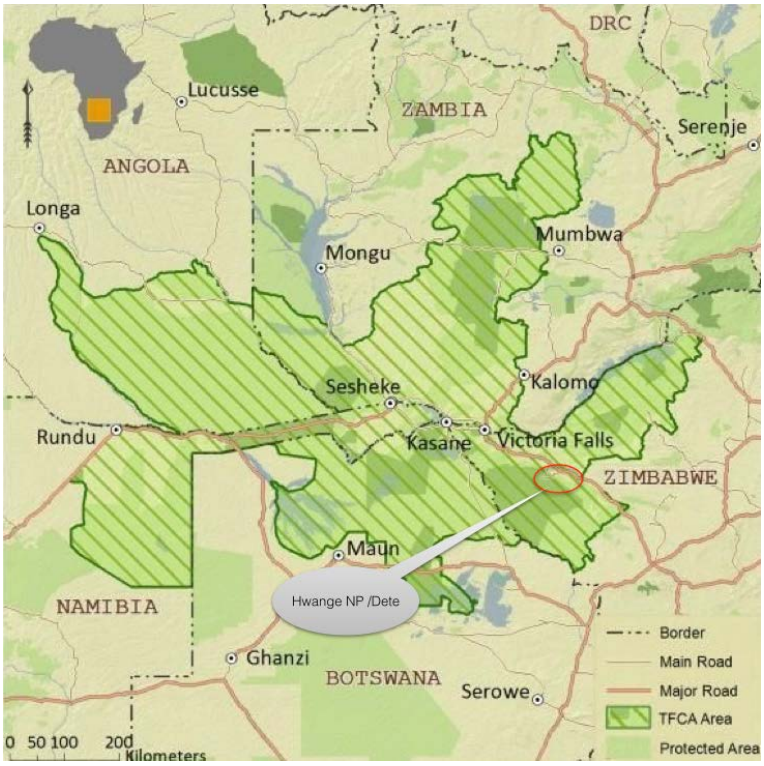
n = 1050 citations. Perceived involvement of wildlife: "Don't know", "No" and "Yes" refers to farmers' replies to the question "Is this disease related to wildlife?" (NA = not available). "Species citation" refers to the number of time a wildlife species has been cited as playing a role in the epidemiology of the disease, and names of often most cited wildlife species (percentage of "wildlife citation")(de Garine-Wichatitksy et al. 2013c).

Studying livestock diseases and how they link with wildlife pathogens can contribute therefore significantly to small-scale farmers health (well-being and livelihoods) and promote a sustainable coexistence between Man & Nature.

1. Three wildlife/livestock interfaces in two TFCAs

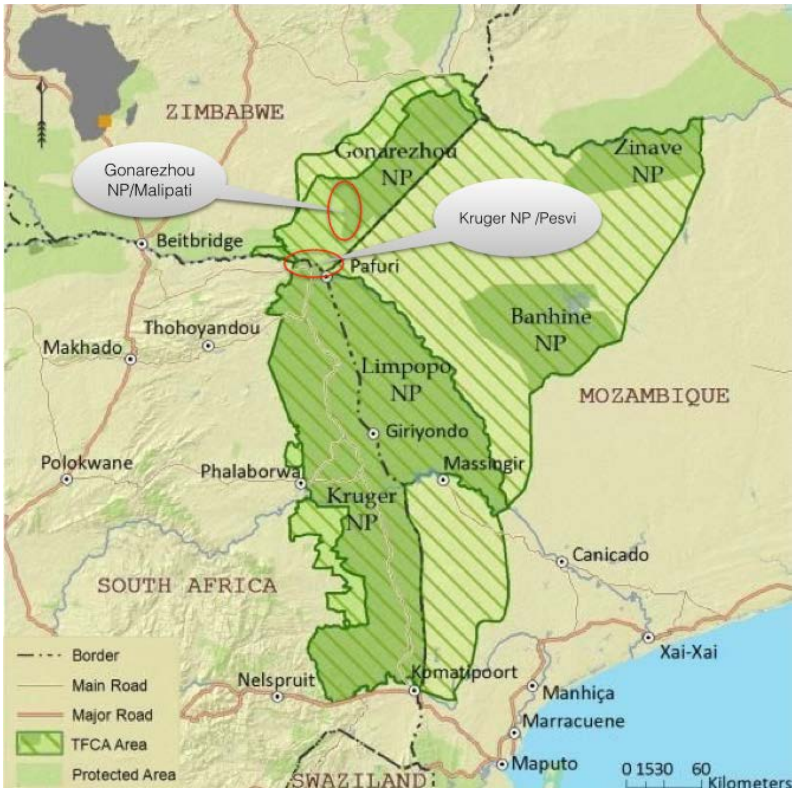
The AGIRs team involved in the work related here is mainly (but not exhaustively) composed of: Michel de Garine-Wichatitsky, Daniel Cornelis, Eve Miguel and Vladimir Grosbois. A CNRS-Cirad team has been involved in the Hwange ecosystem since the end of the 90s and this first SES constituted a logical study site for the study of wildlife/livestock interfaces (Figure 29). Since 2012, this site is located in the Kavango-Zambezi TFCA regrouping parts of Angola, Botswana, Namibia, Zambia and Zimbabwe. We also started in 2007 to work in the Great Limpopo TFCA, the first TFCA signed in 2002 by the presidents of Mozambique, South Africa and Zimbabwe (Figure 30).

Figure 29: KAZA TFCA and the studied interface



In the KAZA TFCA, the studied interface is called the Hwange/Dete interface referring to the Hwange National Park, the largest NP in Zimbabwe and to Dete village, one of the villages in the communal land surrounding the NP. Other villages where we have worked are called Mabale, Sialwindi and Chezhou. In-between the NP and some of these villages, lays the Forestry Commission Area, a protected area where some human activities are tolerated (e.g. dead wood collection; grazing in the first kilometres) and where wildlife roam freely.

Figure 30: GLTFCA and two of the studied interface



In the GLTFCA, we focused on two interfaces. The first one called the Gonarezhou/Malipati interface separating the Gonarezhou NP, the second largest park in Zimbabwe and the Malipati village in the Sengwe Communal Land. Other villages where we have worked are called Palhela, Chikombedzi (20kms from the park). The second interface in the GLTFCA, called the Kruger/Pesvi interface separates the Kruger NP in South Africa, the largest NP in Africa and Pesvi a village in the Sengwe Communal Land in Zimbabwe.

We investigated disease burden, pathogen spread and contacts at wildlife/livestock interface in these 3 interfaces in order to be able to compare our variables across different ecosystems.

2. Emerging pathogens and disease burden at the wildlife/livestock interface

Selection of pathogens, diseases and hosts

Our initial objective (notably through the PARSEL project) was to assess the disease risk and the pathogen load at the wildlife/livestock interface in the GLTFCA and to investigate the role of different types of wildlife/livestock interfaces in shaping the disease burden and pathogen community of cattle and buffalo hosts. Our main animal model has been domestic cattle and African buffalo.

The main reasons for this are:

- Cattle are key socio-cultural assets for small-scale farmers in this semi-arid area and impacts people's livelihoods.

- Buffalo is one of the Big Five for trophy hunting (hence an economic asset) and an important species for tourism as well.
- Both are grazers and represent important large ungulate species for rangeland management.
- They are phylogenetically close and therefore pathogens are likely to be able to infect one of them if they can infect the other.

Those studies spanned over 8 years and covered what seem to be the most important diseases in the area. However, those diseases can be classified in different categories:

- Diseases important for and known by small-scale farmers (e.g. Tick-borne diseases)
- Diseases important for but unknown by small-scale farmers (e.g. brucellosis)
- Diseases of economic importance for trade (e.g. foot and mouth disease – FMD)
- Diseases of zoonotic importance for human populations (e.g. bovine tuberculosis).
- Diseases of importance for the international community but locally of unknown impact (e.g. Rift Valley Fever – RVF)

Table 12: Selected pathogens and associated diseases

Pathogen	Disease	Reservoir	Transmission	Impact	Category
Brucella sp.	Brucellosis	buffalo?	abortion product	Abortion	Zoonosis; Local production
Mycobacterium bovis	Bovine tuberculosis	buffalo, cattle	Environmental; Direct contact	Morbidity Mortality	Zoonosis; Local Production
Theleria parva	Theleriosis	buffalo	Vector-borne	Mortality	Local production
Rift Valley Fever virus	RVF	Unknown (Mosquito population?)	Vector-borne; Direct contact?	Mortality	Zoonosis; Local production
Foot and mouth disease virus	FMD	buffalo, cattle	Direct contact; Environmental?	Morbidity	International trade
Lumpy Skin Disease virus	Lumpy Skin Disease	Unknown	Vector-borne? Direct contact?	Morbidity Mortality	Local production

(Caron et al. 2013)

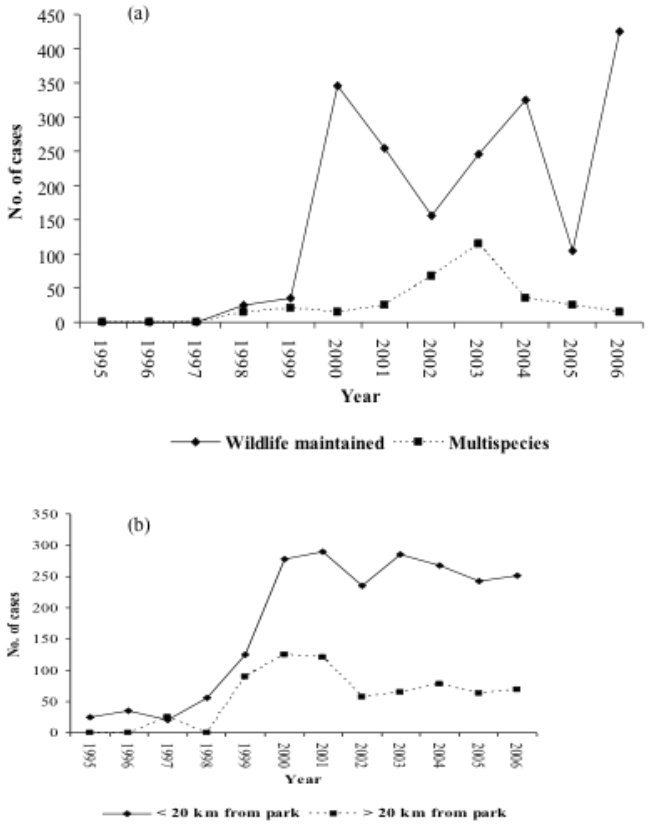
A disease can belong to several of these categories (Table 12). For example, bovine tuberculosis is an emerging pathogen in Zimbabwe, potentially impacting livestock production if endemic and potentially threatening human health if transmitted from animal to human through direct contacts or consumption (i.e. domestic or bush meat).

Concerns of various stakeholders will vary depending on how much they feel concerned by the disease addressed. This is very important especially when Veterinary Services staff "needs" an intervention such as vaccination against FMD and farmers perceive this intervention as useless because they think FMD is not an important disease for their livestock. However, they would like Veterinary Services to get more involved in the control of tick-borne diseases (TBDs) triggering high levels of mortality and morbidity in their herds.

Animal disease burden in the TFCAs

Animal disease is often assumed to be a reality when strong Human/livestock/wildlife interfaces occur. However, clear demonstration of this process seldom exists. One can use Veterinary Services records to investigate patterns of Human/Wildlife conflict. We did this for the Hwange District for the period 1995-2006 and related outbreaks of diseases with diseases with a known ecology related to wildlife species present in the NP (Figure 31a) and with the distance from occurrence locality and border with the NP (Figure 31b)(de Garine-Wichatitksy et al. 2013b).

Figure 31: Annual occurrence of wildlife-maintained and multi-species diseases in livestock during the period 1995-2006, Hwange District Zimbabwe



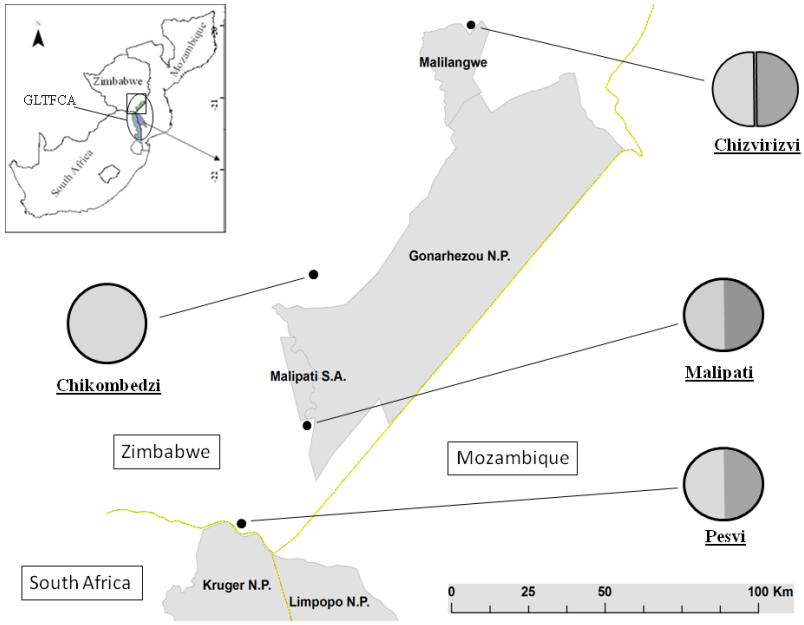
(de Garine-Wichatitksy *et al.* 2013b)

Both these figures indicate that the number of cases in cattle related to a pathogen maintained in wildlife was increasing in recent years and that these cases increased when the village was closer to the NP. One explanation for these observations is that the collapse of veterinary services in Zimbabwe in the 2000s due to the socio-economic crisis led to worse management of diseases at the interface and to an increase in wildlife-maintained diseases, especially close to the NP boundary.

I have collected more recent data in this ecosystem with Last Hungwe, a veterinary student from the veterinary faculty of UZ in 2013 that did his last year project with Cirad. The analyses are not yet available but we tried to relate when possible zoonosis occurrence and outbreaks in human clinics. However, in these settings, diagnostics facilities are so weak in clinics that it is very difficult to link

syndromes with a definitive aetiology. However, this could be the focus of future research.

Figure 32: Study sites and different wildlife/livestock interfaces



The grey area represents protected areas, Malilangwe is a conservancy and Malipati S.A. refers to the Malipati Safari Area, a hunting concession. The single line represents international borders. Each village representing a sampling unit in the study is indicated by a black dot and the circle linked to this dot refers to the type of wildlife/livestock interface: light grey represents livestock and dark grey represents wildlife; the double vertical line separating the circle indicates a fenced interface and a difference in level of shading represents an interface with no fence (Caron et al. 2013).

In 2008-2009, during the PARSEL project, we had an opportunity to implement a protocol to investigate further the relationship between the type of wildlife/livestock interface and the pathogen burden in buffalo and cattle (Caron et al. 2013). In the GLTFCA we selected 4 sites with hard to soft wildlife/livestock interfaces (Figure 32). We sampled cattle populations at the four interfaces and buffalo populations at the two open interfaces (Gonarezhou/Malipati and Kruger/Pesvi) at several occasion between 2008 and 2013. Table 13 presents the main results synthetized from different publications and unpublished data (Gomo et al. 2012a; Caron et al. 2013; Caron et al. 2015b).

Our results indicate that there is a shared burden of pathogens between sympatric buffalo and cattle populations at the different interfaces. Infection does not result systematically in disease (e.g. an infected reservoir host does not develop disease), but it represents evidence of the transmission of a pathogen to the host. The results presented here do not offer proof of inter-species pathogen transmission. They provide a first screening of important infections in cattle populations at different wildlife/livestock interfaces that requires further investigation to understand the pathogen dynamics at play in these multi-host systems. The current knowledge about disease transmission at wildlife/livestock interfaces is still scarce. The definite proof of pathogen transmission in situ at these interfaces is technically difficult.

At soft interfaces, cattle populations share more space and potential contacts with wildlife than with other cattle populations in other villages (Murwira et al. 2013). At the time of the study, limited market opportunities due to national economic instability and minimal transport facilities on tough dirt roads significantly curbed cattle exchanges between villages, even when the distance between villages was only a few dozen kilometres (Giller et al. 2013). The cattle populations in each village thus could be considered to be epidemiological units that are more or less exposed to wildlife and loosely connected to other cattle populations from distant villages.

These results suggest (but do not demonstrate) that the risk of disease spread from one side of the interface to the other varies among pathogens. Other factors could explain the differences observed such as cattle to cattle transmission between villages. However, the present study confirms that the buffalo population could represent a risk of cattle infection by bTB, FMD, theileriosis, as suggested by the literature. On the other hand, cattle could represent a risk for buffalo for Br and hypothetically RVF and LSD if results of this study are confirmed.

This heterogeneity of the sanitary risk across the interface can be explained by the different modes of transmission of pathogens considered. Bovine tuberculosis can be transmitted by direct or indirect contact between hosts (Tanner & Michel 1999). The use of common water holes or grazing areas by buffalo and cattle in unfenced interfaces could result in inter-species transmission of the disease. Cattle owners in Pesvi reported seeing their cattle grazing with buffalo, indicating that direct inter-species contacts are possible at unfenced interfaces.

Table 13: Results for pathogen diagnostics at different buffalo/cattle interfaces

Interface	Soft interface				No interface	Hard interface
Site	*GNP/Malipati*		*KNP/Pesvi*		*Chikombedzi*	*Chizvirizvi*
Host	*cattle*	*buffalo*	*cattle*	*buffalo*	*cattle*	*cattle*
Year of sampling	*2008-09*	*2008*	*2008*	*2010-11*	*2009*	*2009*
Brucella sp.	9.6%	0.0%	16.0%	15.7%	10.5%	0.0%
Mycobacterium bovis	1.0%	10.0%	1.7%	10.4%	0.0%	1.7%
Theileria parva	3.2%	88.2%	42.5%	97.9%	0.0%	0.0%
Rift Valley Fever	18.3%	5.3%	n.a.	5.6%	7.7%	8.5%
FMD	10.0%	94.7%	n.a.	97.8%	13.0%	6.7%
SAT 1	7.1%	92.1%	n.a.	94.5%	13.0%	3.3%
SAT 2	1.4%	68.4%	n.a.	93.4%	5.6%	6.7%
SAT 3	2.9%	65.8%	n.a.	94.5%	3.7%	3.3%
Lumpy Skin Disease	52.2%	0.0%	n.a.	n.a.	48.1%	54.2%

Mycobacterium bovis: SCITT, Single comparative intradermal tuberculin skin test; n.a., not available*; Brucella sp.*:RBT, Rose Bengal test; positivity is decided upon consideration of positivity for both Rose Bengal and c-ELISA tests; *Theileria parva:* IFA, immunofluorescent assay & rPCR, real-time polymerase chain reaction; **Lumpy Skin Disease**: VNT, virus neutralization test. Bovine tuberculosis SCITT results aggregated across the study period. Foot-and-mouth and Rift Valley fever and lumpy skin disease results are given for the October 2008. Brucellosis and theileriosis results are given for samples collected between August 2007 and October 2009 (Caron et al. 2013).

In southern Africa, FMD is transmitted exclusively by direct contact, the hot environment precluding the long distance transmission that can take place in Europe (Thomson, Vosloo & Bastos 2003). Direct contacts between buffalo and cattle were only possible at the two unfenced interfaces. Fences limit the spread of FMD viruses from buffalo to cattle by constraining host mobility, although other wild ungulate species have been involved in FMD transmission (Hargreaves et al. 2004). Interspecific transmission of brucellosis requires close contact between a naive individual and abortion products within a few hours after the latter are dropped on the ground (Gomo et al. 2012a). The use of a shared habitat may result in brucellosis transmission (Nishi, Stephen & Elkin 2002). However, cattle herding and management strategies (e.g. cattle kraaled at night) may result in different temporal patterns of habitat use and thus asymmetric risks of brucellosis transmission. The different modes of transmission between bTB and brucellosis and the type of buffalo/buffalo interaction between GNP and KNP could explain the different patterns of occurrence observed for both infections across the interface. This hypothesis could conciliate apparently contradictory transmission hypotheses previously presented for bTB and Br.

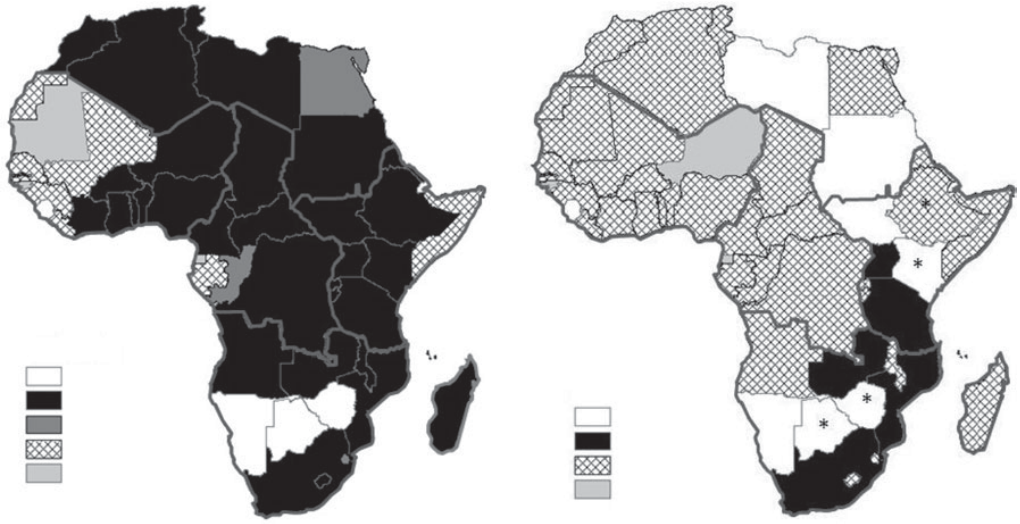
Theileriosis, RVF and LSD are all vector-borne diseases, the former is transmitted by ticks and the latter two by mosquitoes. Vectors are restricted to specific habitats and have limited movement capacities compared to their hosts. However, as long as vectors and wild and domestic hosts share common habitats, even at different times, the transmission of vector-borne diseases may occur. A fence will not limit the transmission of mosquito-borne diseases at the wildlife/livestock interface as mosquitos can fly towards animals across the barrier. Therefore, management of these vector-borne diseases should concentrate on vector control or immunization of livestock. However, a fence could limit the spread of some ticks quite effectively, particularly those that feed on large ungulates, as ticks rely on animals to move them from one place to another. The vectors of buffalo-derived *T. parva* infection, *Rhipicephalus appendiculatus* and *Rhipicephalus Zambeziensis*, are monotropic ticks which feed mainly on large domestic (cattle) and wild ungulates. Large ungulates can neither go through an intact game fence nor jump over it (with some exceptions see (Hargreaves et al. 2004). It therefore is possible to control cattle theileriosis using game fences. The intact game fences in the Chizviridzi area can account for the absence of detection of *T. parva*.

In the following sections, I present some additional work implemented by our team and some RP-PCP students I have been involved with on specific diseases at the wildlife/livestock interfaces and develop two case studies that we have more explored on bTB ad FMD.

Bovine tuberculosis spreading in the GLTFCA

Bovine tuberculosis is an interesting disease regarding not only its impact on cattle production and the role that wildlife can play in its ecology but also in regards to the difference in terms of perception by farmers and veterinary services across Africa (de Garine-Wichatitksy et al. 2013a; Caron, de Garine-Wichatitsky & Roger 2014).

Figure 33: Distribution map of bTB in Africa (1996-2011)



Left: Cattle status at country level; Right: wildlife status at country level. White = Absence of bTB; Black = clinical disease; dashed = No data; grey = Suspected. Asterisk (*) indicates countries (i.e. Botswana, Ethiopia, Kenya, Zimbabwe) where suspected and confirmed cases have been detected but not yet reported to OIE. No additional information (e.g. species) was available for suspected cases reported in wildlife for Niger, Equatorial Guinea and Guinea-Bissau and confirmed cases in wildlife in Mozambique (de Garine-Wichatitksy *et al.* 2013a).

Bovine TB is widespread in livestock on the African continent (Figure 33)(Ayele et al. 2004), whereas bTB infection of wildlife was historically limited to a few countries in southern and eastern Africa (South Africa, Tanzania, Uganda, Zambia). Although the impact of bTB may be significant in Africa, affecting the economy, the ecosystem and human health (Michel, Müller & van Helden 2009), wildlife TB is currently not considered a priority for most African countries. *M. bovis* has been isolated from a wide range of wild free-ranging mammals, including maintenance hosts in natural conditions, which will considerably complicate future bTB control programmes (De Lisle et al. 2002), and possibly compromise the chances of eradication in sub-Saharan African countries. However to date, only one case demonstrating *M. bovis* spillback from infected wildlife to cattle or spillover to human populations have been recently documented in sub-Saharan Africa (Musoke et al. 2015). Currently, bTB in African wildlife South of the Sahara is essentially a conservation issue, with social, welfare and economic implications for the affected areas/countries and not the other way round (Caron, de Garine-Wichatitsky & Roger 2014). Although the ecological impacts of bTB on infected free-ranging populations is still not clearly demonstrated (even for buffalo (Caron, Cross & du Toit 2003) or lions), there is concern in southern Africa that transboundary animal diseases spread by wildlife, and bTB in particular, may negatively impact on economic activities and livelihoods of local people, especially in the context of TFCAs (Michel et al. 2006). In other parts of Africa, it is likely that the risk of bTB transmission between wildlife and livestock (and possibly humans) will increase in the near future, with ever-increasing human encroachment into wildlife habitats due to the demand for grazing and cropping land. This will have serious consequences for biodiversity conservation, as bTB may potentially further threaten several small and isolated wildlife populations (including endangered species at a local or global scale), and also potentially for

veterinary and public health as the wildlife maintenance host will complicate future bTB control or eradication programmes (Kock et al. 2014).

During our initial sampling of buffalos in 2008, we detected bTB for the first time in buffalo in Zimbabwe (Table 13, Figure 34), with further molecular analyses demonstrating that the strain was coming from the buffalo population in KNP (de Garine-Wichatitsky et al. 2010). In 2009, ten buffalos that had tested negative during the initial sampling in 2008 were re-sampled and one tested positive to IFG test, indicating the spread of the disease. The confirmation of results for bovine TB–infected buffaloes in GNP raises several questions regarding the transboundary spread of animal diseases and has considerable management implications for the GLTFCA.

In cattle, even if the SCITT prevalence was significantly different from zero when all cattle samples were combined (Table 13), no SCITT positive cattle were confirmed positive with the IFG (Interferon gamma) test at post-mortem examinations and with lymph node cultures (the golden standard for bTB) (Tschopp et al. 2010). Therefore, the presence of bTB could not be confirmed in the cattle population sampled in our study. Those cattle populations are therefore at risk of bTB spread from the GNP buffalo population. This absence of confirmation of bTB in cattle supports the hypothesis that bTB has been introduced only recently in the buffalo population and identifies a risk of emergence of bTB in cattle in this area (according to OIE, Zimbabwe has been considered free from bTB in cattle since 1996). The recent confirmation of bTB spread from buffalo to cattle in South Africa (Musoke et al. 2015) across the KNP fence emphasizes the reality of this risk in the GLTFCA.

Figure 34: Spread and risk of spread of bTB in the GLTFCA

Left: grey areas represent protected areas and black lines represent international borders; at-risk buffalo populations are represented as circles; crosses denote bTB-infected populations. Right: grey arrows with a cross represent interfaces with cattle and buffalo outside the park, but where there is no evidence for bTB transmission to date; black arrows show bTB transmission route over time through buffalo populations. There is a risk of bTB spread: (1) to cattle in the Sengwe communal land from the KNP and GNP buffalo populations; (2) to the northern GNP buffalo population from the southern population and subsequently to northern buffalo and cattle populations; (3) to buffalo and cattle populations in Mozambique. So far, bTB surveys have not detected any positivity in cattle and buffalo populations in contact with infected populations except as mentioned in South Africa on the western border of KNP (not displayed here)(Kock *et al.* 2014).

The most likely scenario to explain the spread of bTB from KNP to GNP buffalo populations is buffalo to buffalo contact across the boundary because the bTB cases reported here were located <45 km from the unfenced northern boundary of KNP. However, >12 wild species in Kruger have now been found to be infected by bTB (Michel et al. 2006). Most of these species are probably not effective sources of *M. bovis* infection for buffaloes, but the disease epidemiology could rely on multi-host maintenance (Renwick, White & Bengis 2007). Thus, a second scenario could be a buffalo-to–unidentified wild species–to-buffalo pathway, because species like greater kudu (*Tragelaphus strepsiceros*) appear to be able to maintain, spread, and even drive a bTB epidemic (Keet et al. 2001; Michel et al. 2009). A third scenario involves movement of infected livestock across the boundaries of the 3 countries of the GLTFCA, resulting in cattle-to-buffalo transmission of bTB. As a last scenario, we cannot rule out the possibility that bTB infection of buffaloes has remained silent and undetected for decades in Zimbabwe.

The management implications of bTB in buffaloes in GNP are considerable. Once bTB is established in a native free-ranging maintenance host, eradication is unlikely (De Lisle et al. 2002; Michel, Müller & van Helden 2009). Adequate risk-mitigation strategies should be developed and implemented to reduce the risk for bTB transmission to livestock and humans living at the periphery of the unfenced GNP. *Mycobacteria bovis* can remain in milk and soar milk for some days and in meat as well putting rural populations at risk of diseases (Etter et al. 2006; Michel et al. 2015). Bovine TB prevalence in buffalo in southern KNP, initially introduced by cattle, has reached 35-40% (Rodwell et al. 2001) and seems to have stabilized (Figure 34). It therefore is likely that bTB prevalence in buffalo in GNP will increase in the coming years. Although eradication of bTB is unlikely when a wild maintenance host is infected (De Lisle et al. 2002; Michel et al. 2006), a mitigation strategy should be developed and implemented to reduce the likelihood and impacts of bTB spreading to other wildlife maintenance host in GNP, cattle populations and eventually humans in and around the park.

In order to inform decisions on these mitigation strategies, a better knowledge on the routes of transmission between hosts is necessary. We therefore investigated further the mechanisms of spread of bTB from KNP and GNP using telemetry protocols at buffalo and cattle interfaces (see below).

Foot and mouth disease in southern Africa, myths and reality

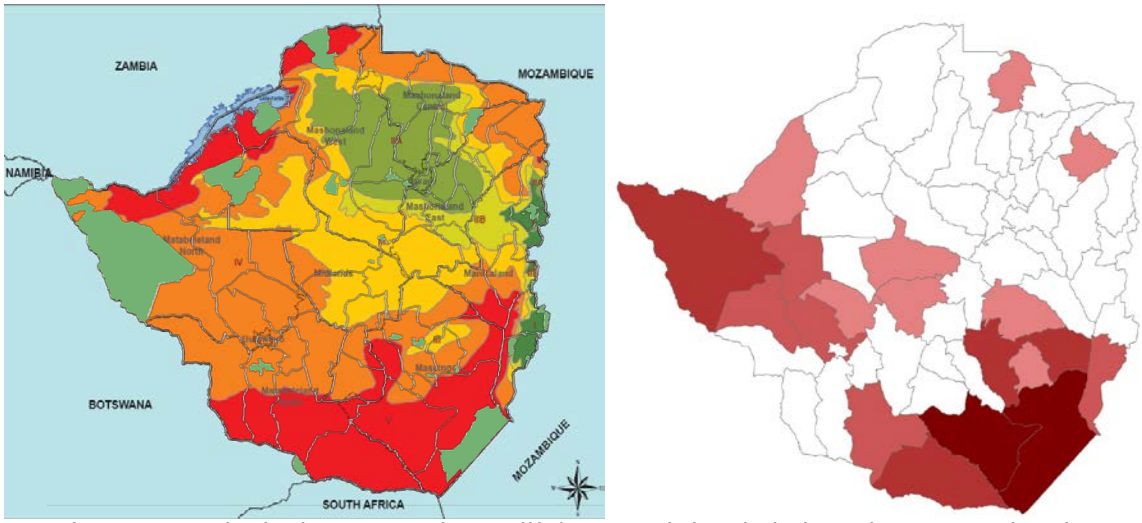
The control of FMD in southern Africa is at the heart of an intense debate between departments of veterinary services (DVS) and conservationists (this is not as Manichean as that and there are many other stakeholders involved in this debate). The former wants to segregate infected zones from disease-free zones in order to be able to access lucrative meat export markets such as the European Commission market. South Africa, Namibia, Botswana and in the 90s Zimbabwe erected thousands of kilometres of fences around their NPs where the African buffalo, the maintenance host of the virus, lives (Ferguson & Hanks 2010; FAO & OIE 2011). The later see fences as a constraint to animal movements and ecological connectivity within landscapes, threatening biological processes and the conservation of biodiversity (Cumming et al. 2015a). In addition, farmer communities living in the periphery of these NPs are taken hostage of FMD control, as they constitute a buffer of FMD surveillance to control for FMD circulation in their area. As a consequence, their livestock trade options are very limited. FMD is therefore an important disease in southern Africa (which is not the case in the rest of Africa) and current initiatives to which I have (modestly) contributed are blooming to reconcile the development of peripheral communities and conservation of biodiversity (Ferguson et al. 2013; Thomson et al. 2013a; Thomson et al. 2013b).

The understanding of FMD ecology in southern Africa (which so far dictates the dogma for the rest of Africa) is that: 1) it is maintained in buffalo populations; 2) it can be maintained in cattle population but veterinary control usually prevent this situation; 3) the role of other wildlife hosts is largely unknown; 4) FMD is transmitted by direct contact only and a double-fence separated by a few meters should be enough to prevent transmission; 5) the virus cannot survive in the environment because the climatic conditions are to harsh, preventing any environmental transmission; 6) FMD topotypes (SAT 1, 2 &3) occur in southern

Africa with some geographical and host clustering (Vosloo et al. 2002; Thomson, Vosloo & Bastos 2003; Vosloo et al. 2005).

With some Zimbabwean colleagues, we managed to access a database of FMD outbreaks in Zimbabwe from 1931-2006. After some work with colleagues from the DVS, we identified the FMD primary outbreaks within this database and analyse their clustering at the district level (Figure 35).

Figure 35: Agro-ecological zones & FMD primary outbreaks per districts in Zimbabwe (1931-2006)



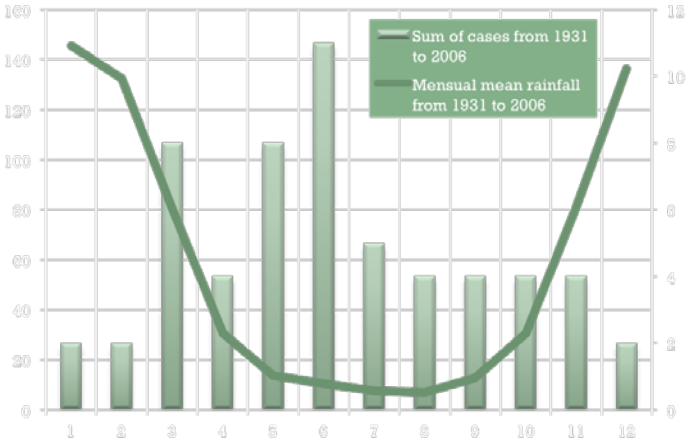
Left: Agro-ecological zones and NPs (light green) in Zimbabwe (orange and red zones are semi-arid areas); Right: Primary outbreaks per district in Zimbabwe (the darker the higher number of FMD outbreaks)(Guerrini *et al.* In prep).

The comparison between the two maps indicates that primary FMD cases occur in districts where the two largest NPs in Zimbabwe occur (i.e. Hwange and Gonarezhou NP). Of course these areas correspond to the habitat of the largest buffalo populations in Zimbabwe and to potential wildlife/livestock interfaces. A cluster analysis (using SaTScan) implemented by Laure Guerrini indicated that a significant cluster existed only in the South East Lowveld of Zimbabwe around the Gonarezhou NP. Looking at this area in particularly, we could demonstrate a clear relationship between rainfall and FMD primary outbreaks (Figure 36).

From these analyses, we have designed a hypothetic model of FMD transmission at wildlife/livestock interfaces and risk for FMD outbreaks in cattle populations. The increasing incidence of outbreaks between March and June corresponds to the drying of non-perennial water points in NPs and interface areas (Figure 36). This progressive drying-up of water points leaves ungulates relying on natural surface water to progressively use more and more perennial water points. The more and more scarce surface water (e.g. water pools in river bed) attracts therefore both domestic and wild ungulates and these increasing contacts between cattle and buffalo can lead to more potential FMD infectious contacts and hence, primary outbreaks in cattle populations (Figure 37). Alternatively, environmental conditions during the cold-dry season in this semi-arid area could favour the survival of FMD virus in the environment and promote indirect transmission (a hypothesis against current FMD dogma in southern Africa). Both hypotheses are not exclusive. The March to June window is quite wide but in these semi-arid areas, a high annual variation in rainfall patterns can lead to shifts in the timing of non-perennial water

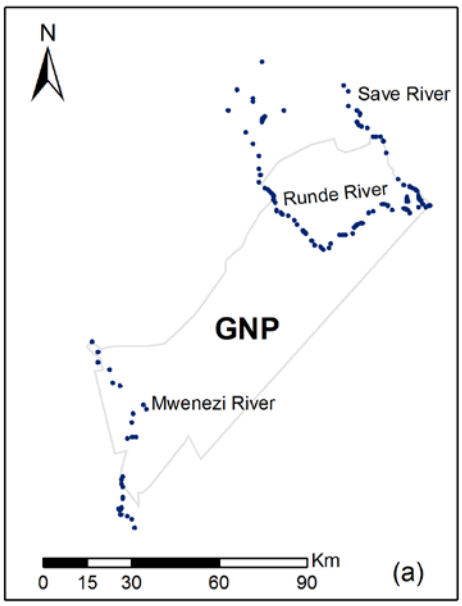
points drying-up. We will investigate this hypothesis in a manuscript in preparation (Guerrini et al. In prep).

Figure 36: Relationship between rainfall and FMD primary outbreaks in the South-East Lowveld of Zimbabwe



Each bar represent 1 month of FMD primary outbreaks starting in January; the line represent average rainfall (Guerrini *et al*. In prep).

Figure 37: Water point available during a particularly dry season (2005) in the GNP



Zvidzai, Murwira, Ndaimani, Caron & de Garine-Wichatitsky, unpublished

The presence of antibodies in the cattle populations tested (Table 13), in the absence of recent vaccination against FMD (Governmental Veterinary Services, personal communication), indicated a recent circulation of FMD virus in cattle in 2008 and 2009 (no information on the topotypes circulating could be inferred from serological results). During an in situ vaccination trial implemented for the CORUS FMD project, regular sampling and FMD testing of a control group of cattle in the periphery of GNP during 10 months in 2010 indicated again an on-going circulation of FMD viruses with probable spillover at the wildlife/livestock interface in August-November or later early during the rainy season (Jori et al. 2014). Follow-up

serological survey reported also FMD circulation in 2011 in the cattle population in this area during the dry season (Miguel et al. 2013b). This cumulative evidence of FMD circulation in cattle population of Zimbabwe in the GLTFCA proves the recurrent exposure of cattle to buffalo FMD strains. The timing of the estimated FMD transmission between buffalo and cattle does not match the March to June window that we previously identified. However, this window was calculated on averaged rainfall over 75 years when this recent seroprevalence was observed over 3 years that could have been be wetter than the average, resulting in the drying-up of non-perennial water points later in the year.

Finally, our studies also demonstrated that similar FMD strains were circulating between cattle (in Mozambique) and buffalo (in South Africa and Zimbabwe) in the GLTFCA further supporting the inter-species and transboundary dynamics of FMD strains at wildlife/livestock interface. These strains remained undetected in cattle during our study, raising the risk of invisible circulation of FMD strains in cattle populations (threatening exportation zones). These observations were echoed at the time with a FMD outbreak in South Africa outside its surveillance zone (in the Kwazulu-Natal province), resulting in a ban of meat exportation from South Africa to Europe for close to a year. Last point, the *in situ* vaccination trial that I implemented in Zimbabwe with the trivalent vaccine from the Botswana Veterinary Institute (only available vaccine in the region at the time) resulted in an antibody response of 4 months only, indicating the need but impossibility for financial and practical reasons, to vaccinate these populations 3 times a year to get a proper vaccine coverage.

We strengthened these studies with telemetry studies as shown in a later section.

Other pathogens, other diseases at the wildlife/livestock interface

Mostly through MSc, Mphil and PhD students, we have also investigated other pathogens and diseases at the wildlife/livestock interface.

As mentioned earlier, ticks and tick-borne diseases (TBDs) are important diseases for farmers. In Zimbabwe, we tried from the beginning to engage farmers to collaborate with us on research protocols through the provision of acaracides for diptank. The diptank system in Zimbabwe is the centre of the epidemiosurveillance for DVS and farmers are used to benefit from this service delivery. The impact of TBDs is therefore lesser than in other country with a less developed and organised dipping activity. Nonetheless, TBDs still have an impact on cattle production.

In Table 13, we presented some results on *Theileria parva* with a clear indication of the role of different types of interface on the transmission of the buffalo-derived theileriosis commonly referred to as Corridor Disease (CD). Buffalo is the maintenance host for *T. parva* as shown by our results and cattle to cattle transmission are supposed to be rare for *T. parva*. The absence of *T. parva* antibodies in cattle populations with no interface or with hard interface, coupled with their detection in cattle in Malipati and Pesvi, strongly suggests that the origin of the Theileria infection in cattle is in buffalo. The direct transmission of *T. parva* from buffalo to cattle is associated with CD. Corridor disease usually causes severe mortality in cattle (Uilenberg 1999; Latif et al. 2002). Only a few CD outbreaks in cattle were reported in this region prior to and during the study. These results suggest that contrary to common belief (Sibeko et al. 2008), cattle populations

living in close and constant contact with buffalo may support *T. parva* infection without high mortality. I contributed modestly to 2 studies related to tick communities at the interface and associated TBDs (Miguel et al. 2014; Hove et al. In prep).

Brucellosis (*B. abortus*) was not detected in the 38 GNP buffalos tested in 2008, which contrasts with the detection in KNP buffalo populations and previous studies in other areas of Zimbabwe and southern Africa (Chaparro & al. 1990; Madsen & Anderson 1995). However, brucellosis was detected in all cattle populations except at the fenced interface (Gomo et al. 2012a). The absence of brucellosis in buffalo is counter-intuitive and could be explained by: 1) some cattle herding strategies such as cattle kraaling at night which could reduce the potential for buffalo getting infected from abortion products left in the environment by cattle; 2) a possible isolation of the buffalo population in GNP from other infected buffalo populations such as the KNP buffalo population; 3) a small buffalo sample size that would fail to detect a low prevalence in buffalo. We discussed these hypotheses in the thesis and a paper by a MPhil student (Gomo et al. 2012a) later strengthened by a molecular analysis of the strains isolated in cattle (Gomo et al. 2012b). An on-going collaboration with Terramo University in Italy aims at screening a large dataset of cattle and buffalo sera for brucellosis antibodies in order to be able to look at the dynamics of antibodies across 7 years of sampling and the influence of the different types of soft cattle/buffalo interfaces.

The role of wildlife in the epidemiology of LSD is unclear (Hedger & Hamblin 1983; Bengis, Kock & Fisher 2002). Antibodies in African buffalo for the disease have been found during previous studies (Davies 1981; Davies 1982). However, as LSD is mainly vector-borne diseases, the epidemiology is also dependent on the population dynamics of the mosquito vectors (Chihota et al. 2001). No antibodies were detected in the buffalo population. High prevalence was observed in all cattle populations, which correlates with observed LSD symptoms detected in cattle populations in 2007 (Caron, personal communication). LSD is highly prevalent in cattle in all countries in the GLTFCA and have a high impact on cattle production and welfare (LSD cerates invasive open wounds on the body of the animals) but too little effort is invested in understanding its epidemiology including basic knowledge such as transmission modes (Lubinga et al. 2015). This disease could be the focus of more of my work in the future.

RVF is an emerging disease threatening human and livestock health in Africa and the Middle-East. The role of wildlife in the epidemiology (maintenance and transmission) of the virus at human/livestock/wildlife interfaces in southern Africa remains unclear. In order to elucidate RVF epidemiology in the GLTFCA and KAZA TFCA, we combined preliminary data from serology, entomology, telemetry and field experiments to infer on transmission and persistence mechanisms of RVF virus in Zimbabwe. KAZA TFCA serological results were all negative. In the GLTFCA, the seroprevalence was 4.6% in cattle (27/587), 3% in small ruminants (2/66) and 6.8% in buffaloes (14/205). Statistical analyses showed that the transmission was endemic in livestock. Mosquito-trapping resulted in the capture of 6 species, among which 3 potential RVF vectors: *Culex quinquefasciatus*, *C. theileri* trapped in both sites and during the 3 sessions; Anopheles coustani identified in KAZA TFCA site only. Estimated residence time of foetus ranged between a few hours and several days (using bags of viscera as a proxy). These preliminary results indicate

that RVF circulate in cattle and buffalo populations within the GLTFCA, with significant inter-annual and spatial variations, and that transmission between wild and domestic compartment may be due to both vectors and direct contacts (de Garine-Wichatitksy et al. In prep).

3. Human/livestock/wildlife movements and their drivers in TFCAs

I have exposed in section B the complexity of land-use types in TFCAs and the complex network of interfaces created. In the previous section, I have demonstrated the disease burden on livestock, livestock production, human health and wildlife. The impact on human health is direct through potential zoonoses (little studied so far) and indirect through the impact on livestock production one of the pillar of livelihoods in these semi-arid areas. The impact on wildlife populations is not easy to assess as it was for Rinderpest for example (Caron & Kock 2004; Roeder, Mariner & Kock 2013; Thomson, Fosgate & Penrith 2015) and requires exhaustive and invasive studies (Beechler et al. 2015). However, from a conservation point of view, wildlife health should be protected from the invasion of alien pathogens (such as bTB in KNP and GNP) with a priori unknown consequences.

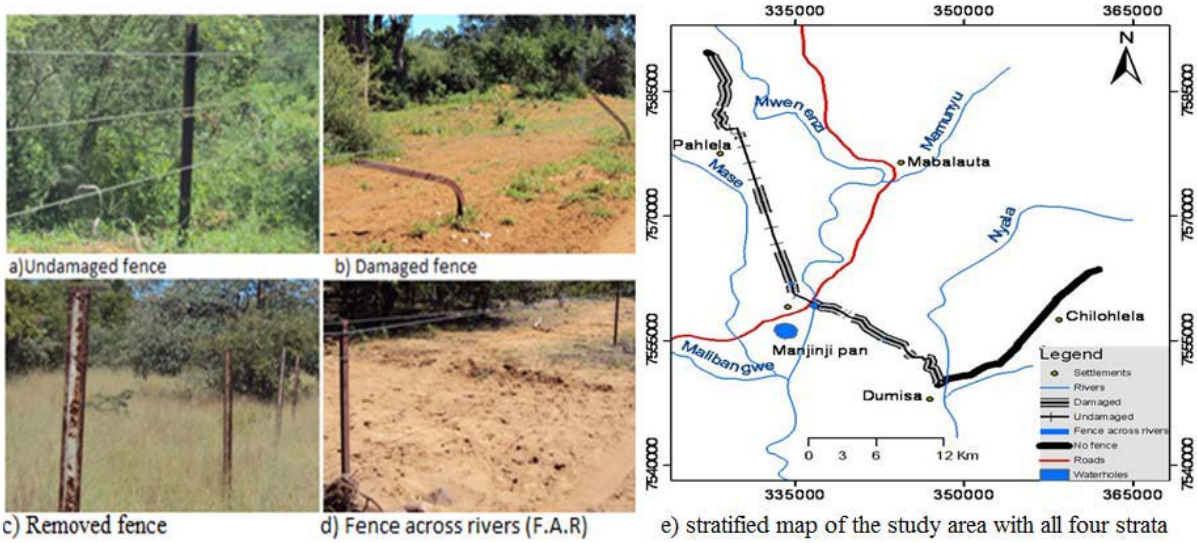
The complexity of wildlife/livestock interfaces requires in order to tackle disease ecology in multi-host systems, a level of understanding of host movements, driving inter-host contacts potentially leading to infectious transmission.

Proxies of host mobility and their drivers

A number of non-invasive techniques can help understand movements and their drivers. A common technique in conservation to collect information on host mobility is to implement road counts (or foot transects) that also inform on host densities when enough data is available. Since the beginning of our activities in the GLTFCA, we implemented regular road counts in the NP, in the interface area and in the communal land (de Garine-Wichatitsky et al., unpublished data). This data gave us an idea of the seasonal frequentation of both land-use types by the different types. This data was completed by formal and informal discussions with farmers (de Garine-Wichatitksy et al. 2013c), discussions and data collected by national park rangers (Murwira et al. 2013) and personal information. These first set of data informed us on a broad pattern on host mobility: wildlife and in particular buffalo do not frequent much outside NPs; cattle do frequent a few kilometres inside GNP, mainly during the dry season (August to November) and to a lesser extent during the months of February and April.

Two MPhil students implemented a study on the veterinary fence (or fence line when the fence was absent) and the use of the fence by wildlife and cattle (Dube et al. 2010; Chigwenhese et al. Submitted after revision) around GNP. On Figure 38, one can see the overall state of the fence damaged by elephant and removed by farmers willing to ensure a safe entry into the park of their cattle.

Figure 38: Four different strata characterizing veterinary fence in the southern part of GNP

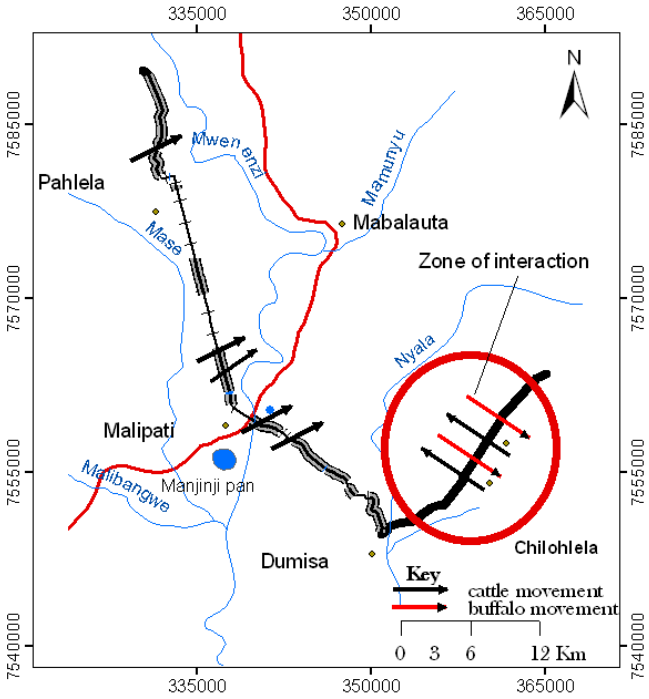


(Dube et al. 2010)

Results of these studies confirm our preliminary observations on the use of cattle and buffalo of "the other side" and bring additional information (Figure 39). Cattle and buffalo do not cross the undamaged fence as expected (this veterinary fence has been erected principally to prevent contacts between cattle and buffalo). Cattle do cross the damaged fence area close to villages (Chigwenhese et al. Submitted after revision) but buffalo mainly cross the fence line when the fence has been completely removed (Dube et al. 2010). We observe a clear seasonality in fence crossing according to the species considered. Buffalo tend to leave the NP during the wet season at fence sections fully removed by elephants; Cattle crossed the fence during the wet season but mostly during the dry season and close to villages where the fences was damaged by humans (de Garine-Wichatitksy et al. 2013b; Murwira et al. 2013).

As mentioned previously (Figure 37), another Mphil student worked on the distribution of surface water in the southern part of GNP. His results clearly indicate that water is a limiting factor during the dry season in the southern part of GNP when the only available surface water remains in a few perennial water points along the Mwenezi River where wildlife and domestic animals come to drink. In addition, water-dependent species such as buffalo (cannot go further than 10kms from water) would not be able to cross from the southern to the northern part of GNP because of the lack of any water point between the 2 main Rivers (Figure 40)(Zvidzai, unpublished data).

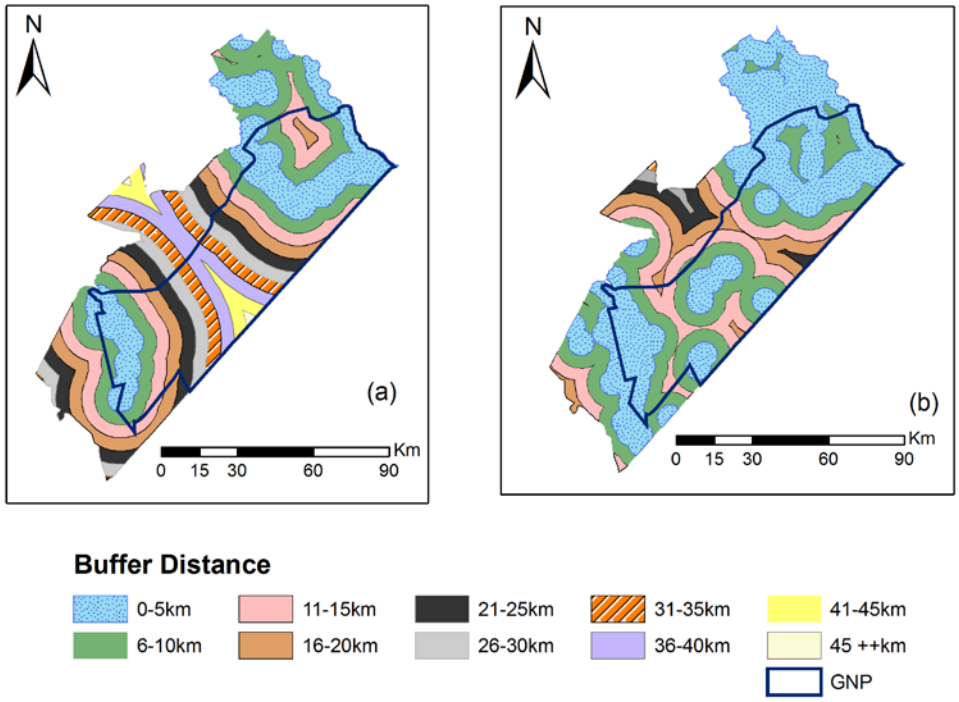
Figure 39: Main locations of crossing by cattle and buffalo in southern west corner of GNP



The zone of interaction indicated with a red circle is not the only one. Obviously, when cattle cross into GNP they can come into contact with buffalo (Dube et al. 2010).

An additional systematic protocol when handling wildlife is to ear-tag them with coloured and numbered tag. This proves important to re-sight individuals within herds and collect additional information on their physical and reproductive status. On one occasion it proves very useful when a wildlife professional sighted buffalos on the outer side of his ranch fence and managed to see a tag, its colour and number; After contacting us, we managed to identify this individual as a young female tagged the previous year in KNP close to 100kms from where it has been sighted in Zimbabwe (See Figure 45 for data). This proves extremely valuable information despite the low probability of accessing such data.

Figure 40: Buffer framework indicating distance to water for different classes



Buffalo need to stay in the first 2 classes (0-10km) in order to go back for drinking on a daily basis. Zvidzai, Murwira, Ndaimani, Caron & de Garine-Wichatitsky, unpublished

This preliminary work gave us some hypotheses to confirm with the telemetry studies that we implemented in parallel. Results from these protocols arrived a couple of years after the start of these studies and it was interesting to have already some knowledge about the global patterns of land use by cattle and buffalo.

Host phylogeny and contacts

Nathalie Smitz is currently finishing her PhD on buffalo genetics. We have contributed in data and inputs to her first two articles (Smitz et al. 2013; Smitz et al. 2014) that reassessed 1) for the first one, buffalo phylogenetics at the species-level, redefining on molecular basis subspecies across the African continent, 2) for the second one, the connectivity at the meta-population level between buffalo population in southern Africa and notably between the KAZA and GL TFCAs. We are expecting soon to get involved in her last chapter for which she has developed a set of SNPs for buffalo and which should be able to give us an index of buffalo population and individual relatedness (i.e. up to the parental relationship between two buffalo hair or tissue samples). With this tool, we will have another way of estimating contact between populations and try to investigate the relationship between relatedness and contact patterns, behaviour and to some extent pathogen transmission.

Telemetry studies at buffalo/cattle interfaces

From the beginning of our work in Zimbabwe, in 2008, with Daniel Cornelis and Michel de Garine-Wichatitsky, we implemented telemetry protocols at wildlife and cattle interfaces. Through the PhD thesis of Eve Miguel, assisted by Vladimir

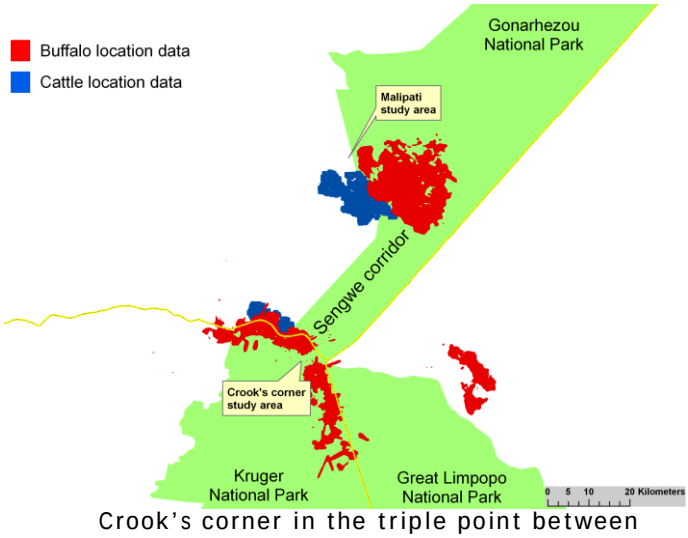
Grosbois, we further continued these protocols and increased our sample size with two extra interfaces between 2010 and 2011. Currently Hugo Valls is finishing is PhD with another dataset on cattle/buffalo interactions in the KAZA TFCA (Valls Fox *et al.* In prep). Altogether, through the projects EU-PARSEL, BUCATIN, ANR FEAR and ANR SAVARID, the team gathered data on 3 buffalo/cattle interfaces in 2 TFCAs across 7 years. This represents an estimation of 90 GPS collars deployed in buffalo and 53 on cattle (Table 14).

Table 14: Synthesis of telemetry studies

	GNP/Malipati	KNP/Pesvi	HNP/Dete	Total
Buffalo	20	50	20	90
Cattle	17	15	20	52
Range	09/08-12/12	06/10-10/15	04/10-12/13	09/08-10-15
Estimated % of success	80	65	75	

As usual with this type of protocol, many failures occurred. For example, in July 2011, out of the 18 collars deployed on buffalo along the Limpopo River at the border between KNP and Sengwe Communal Land in Zimbabwe, the large majority was not anymore functioning after 6 months because of a technical problem. Once replaced by the company, they were fitted again in October 2013. Figure 41 provides an overview of the interfaces that we covered in the GLTFCA.

Figure 41: Overview of the 2 buffalo/cattle interfaces in the GLTFCA



Crook's corner in the triple point between Mozambique, South Africa & Zimbabwe.

The collection of this data represents an enormous amount of work, even if recently with the development of satellite GPS collars, data can be collected directly from the Internet or through cell phone networks. Initially (between 2008 and 2010), data had to be remotely downloaded from the collar with a VHF antenna at a distance of 100 to 300 metres from the animal. If this does not pose any problem with cattle, with buffalo, it requires a lot of fieldwork that I performed during 2 years in the GLTFCA (of course I was not the only one to implement this data collection). At the end, this data proved important to better understand the ecology of buffalo and cattle at this interface and to address the consequences in terms of risk of pathogen transmission and disease spread across these interfaces.

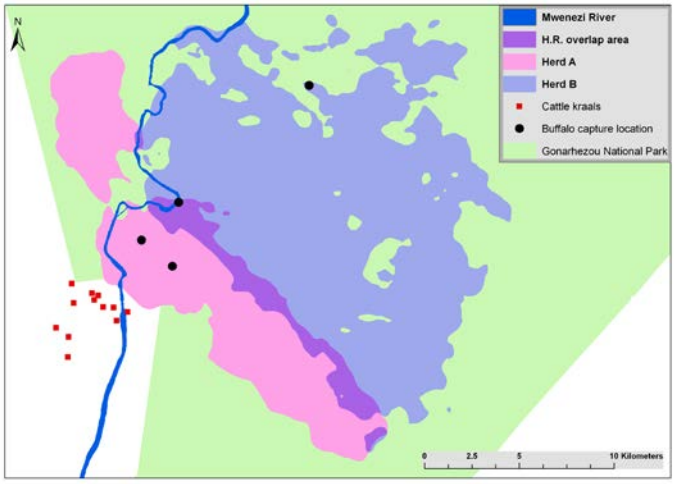
Buffalo & cattle ecology at wildlife/livestock interface

Telemetry data provides information on the location of the individual equipped with the device. The accumulation of this data over time (the GPS collars that we used usually took 1 o 2 GPS locations every hour) informs on the precise whereabouts of the animal within its landscape, relatively to other individuals equipped (from the same or different species), its seasonal preferences for or constraints on resource-use, its use of different land-use types. The development of sensors within collars allows today implementing remote behavioural studies on some species (Benhamou et al. 2014; Elliot et al. 2014). This data can therefore support behavioural ecological studies.

Fadzai Zengeya investigated during her PhD the ecology of cattle in the southern part of GNP. I was not much involved with her published work even if I contributed to the design of her thesis, developed the telemetry protocols and supported extensively the student for the implementation of her research in the field. I will therefore not develop her results here but they have been extensively published (Zengeya, Murwira & de Garine-Wichatitsky 2011; Zengeya, Mutemba & Murwira 2012; Zengeya & Murwira 2014; Zengeya, Murwira & De Garine-Wichatitsky 2014; Zengeya, Murwira & de Garine-Wichatitksy 2015).

It is difficult to assess the proportion of the observed buffalo behaviour that is due to the presence of the interface or simply standard buffalo behaviour that we did not know about. I will develop two aspects of buffalo behaviour that we are currently investigating in a working paper: buffalo herd fusion-fission patterns (Cornelis et al. In prep) and buffalo dispersal between herds (Caron et al. 2015b). The ecology of the African buffalo has been the focus of extensive studies during the last 50 years (Sinclair 1977; Mloszewski 1983; Taylor 1985; Prins 1996). The grouping patterns of this species appear to be well understood: mixed herds gathering adult females, young, juveniles and a smaller proportion of adult males constitute the main ecological units. Bachelor groups gravitate around these mixed herds, joining them mainly for mating and leaving them to escape intra-species and gender competition and improve their resource off-take efficiency until the next mating opportunity (Turner 2005). The mixed herd is defined by: a given home range, with little inter-annual variation and bachelor groups can “migrate” between mixed herds ensuring gene flow between herds (Van Hooft 2003); a static and stable group size often affected by temporary and seasonal fission-fusion patterns (Prins 1989); or by association patterns between individuals (Cross et al. 2004). Figure 42 displays the home ranges (HRs) of 11 adult female buffalos followed between October 2008 and November 2009 in the southern part of GNP and identifies clearly two buffalo herds (A & B) with individuals belonging to the same herds sharing between 65 and 78% of their HRs respectively for the herds B & A. Inter-herd overlap ranged from 3 to 8% depending on the buffalo considered.

Figure 42: Home range of 11 adult female buffalo in GNP

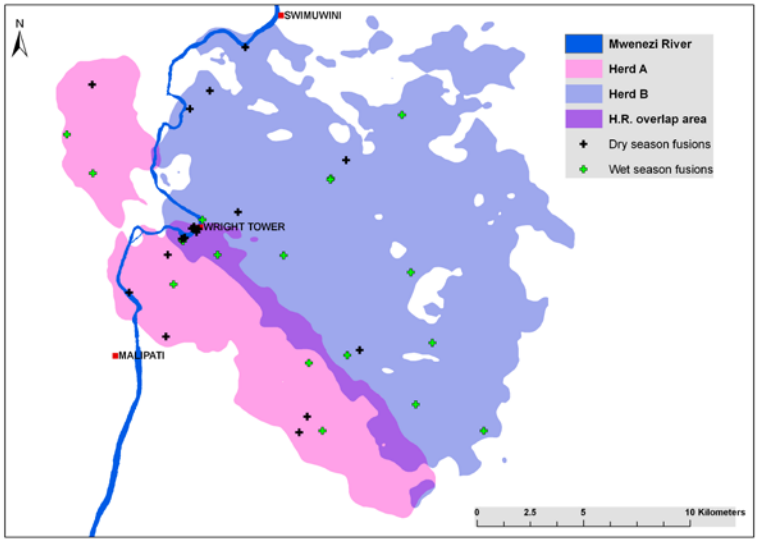


(Cornelis et al. In prep)

However, this well described "planet-satellites"-like framework suffers challenges when some observations systematically question the herd concept as currently perceived. First, home range overlap varies greatly between pairs of herds, challenging the herd definition based on home range only (Hunter 1996; Cornelis et al. 2011). Second, fusion-fission patterns between/within mixed herds occur often, usually seasonally challenging the static and stable properties of mixed herds (Cross et al. 2004; Tambling et al. 2012). Third, group sizes are highly heterogeneous and vary from a few individuals to several thousand (Mloszewski 1983). These observations are systematically reported by all authors and their description is often confused by a vague semantic: two "mixed herds mixing" or a herd being joined by another "group" or "subgroup"; and how do we call the new entity: a "large" or "mega" herd? The complexity to properly identify individual buffalos within large groups and to estimate regularly the size of the group around focal individuals explain the difficulty to understand the group/individual dynamics within herd/groups so far. Recent advances in telemetry technology have permitted more detailed studies that challenge some aspects of the mixed herd concept (this study and (Halley 2002; Cross et al. 2004). Within our study, we observed that at the intra-herd level, buffalo supposed to belong to the same herd were in fact only spending 21 to 44% for herd A and 10 to 22% for herd B of their time together. This means that adult female buffalo that we observed sharing most of their HR and therefore belonging to the same herd spent in fact very little time together. This is contrary to the current definition of a buffalo herd.

In addition, the pairs of adult female buffalo were not stable across time indicating that a number of fusion and fission events occurred through the year. The spatial distribution of those group-level events (here estimated at the pair-level; this is of course a limit to the study) was not distributed randomly in the landscape. The fusion events were strongly related to surface water during the dry season (Figure 43)(Cornelis et al. In prep).

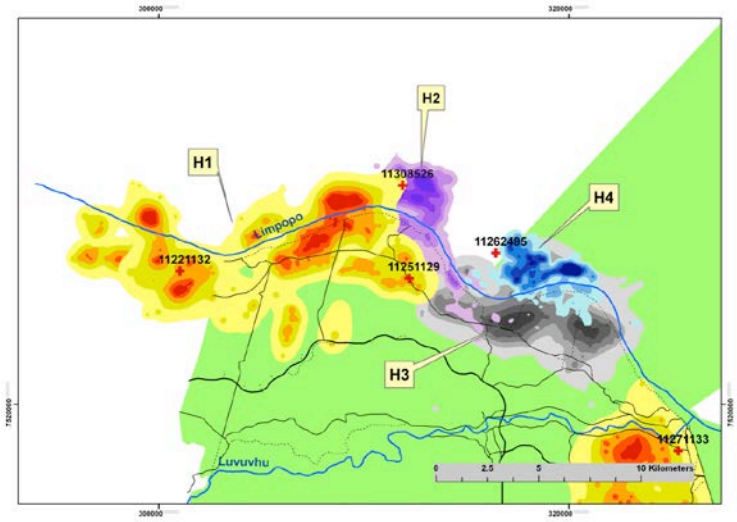
Figure 43: Spatial distribution of fusion events according to seasons



(Cornelis et al. In prep)

In October 2013, we had 18 new buffalo satellite collars to deploy in the KNP/Dete interface with the help of the SANParks capture team (South African National Parks, an excellent collaborator). We decided to test a new hypothesis on buffalo behaviour: the role of young female in linking distant herds. Our numerous observations of adult female HRs (n=47 only in the GLTFCA) led us to understand that their role in connecting distant herds was limited. Their HRs were confined around the water and grazing resource and relatively stable from one year to the next. In the case of the KNP/Dete interface, adult female buffalos had HRs along the Limpopo River, ranging only a few kilometres from the river, in between KNP in South Africa and the Sengwe communal land in Zimbabwe (Figure 44).

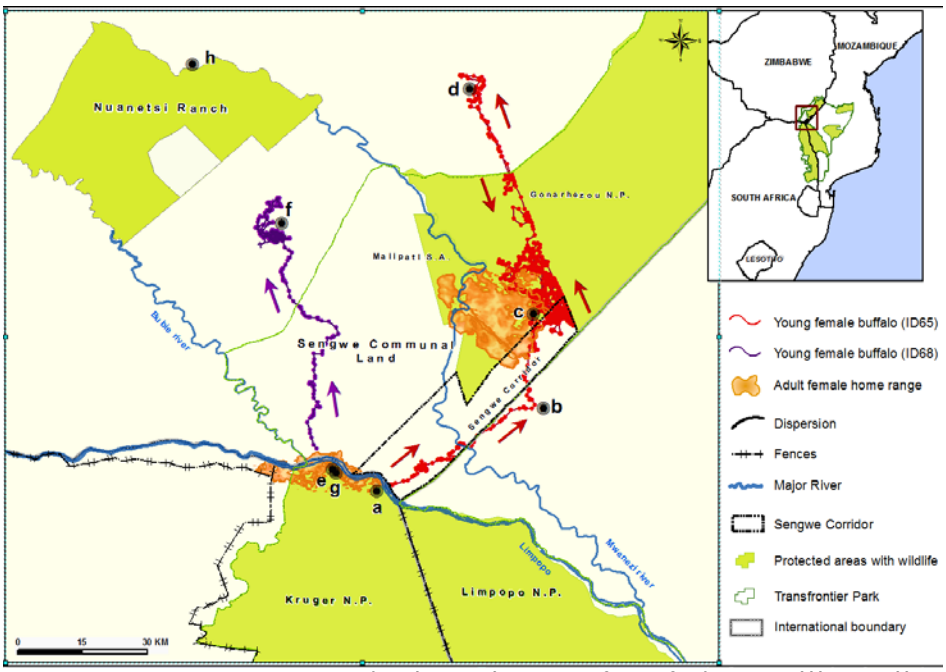
Figure 44: Home ranges of adult female buffalo in the crook's corner (2010-2011)



The Limpopo River separates Zimbabwe (North) from the KNP (green south). The green extension on the right hand side represents the proposed Sengwe Corridor linking KNP and GNP (not gazetted yet)(Cornelis et al. In prep).

However, informal reports by wildlife rangers and veterinarians of young female (between 2.5 and 4.5 years-old, before their first reproduction) dispersing from their native herds and the re-sighting of one of our young female more than 100kms from her capture site a year earlier (see Figure 45, point h), convinced us to equip 19 young females with satellite collars. In a matter of 6 months, 2 of these young female buffalos did disperse from KNP to Zimbabwe covering flight distance of 65 and 95kms in only a few days (Figure 45). In January 2014 and in 6 days, one of them, a 2.5-years old female buffalo crossed into Zimbabwe then Mozambique and in Zimbabwe again to enter GNP, with localizations within the home range of the buffalo herd where bTB was first diagnosed in a female buffalo in 2008 as described in (de Garine-Wichatitsky et al. 2010). She later left the park to visit a commercial farm area before coming back into GNP. Additionally of interest is that 1) one of these young female buffalo left the boundary of the TFCA; 2) the other one used the Sengwe Corridor, a proposed corridor to link KNP and GNP; 3) the 3 events observed occurred during the rainy season.

Figure 45: Two pathways of dispersing young females



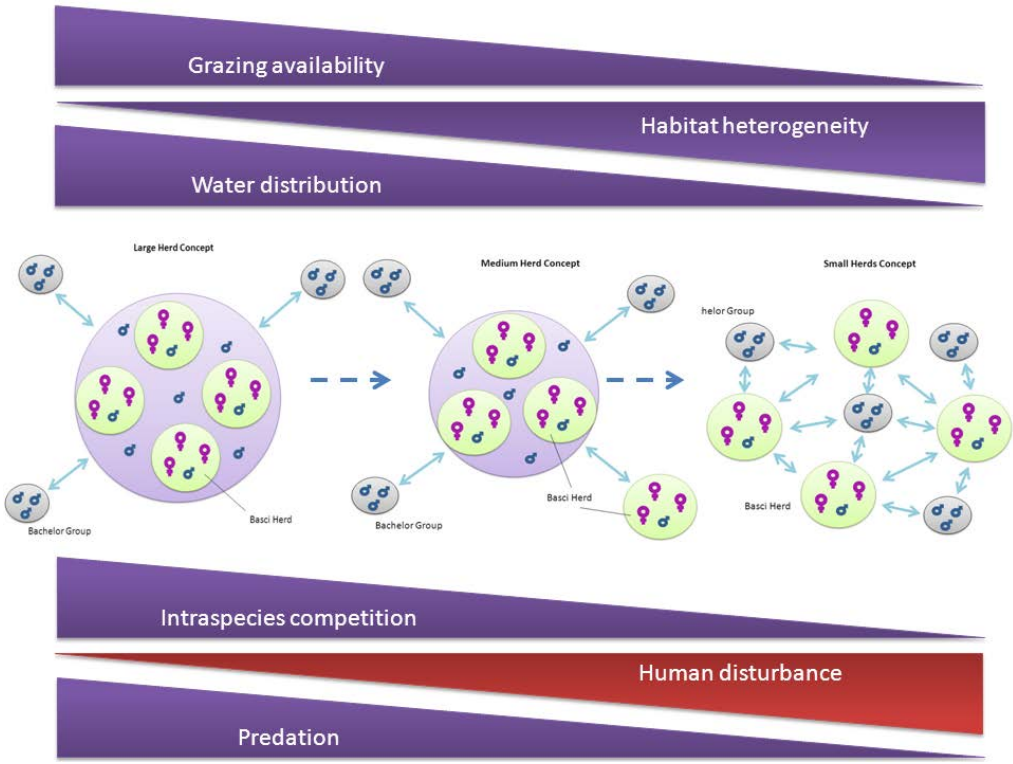
Orange areas represent the intensity HR of 5 of the satellite collar-equipped adult female buffalos, representative of the five herds followed during the study in KNP and GNP. Three cases of long-distance movements of young female buffalos are presented (arrows represent direction of movements): 1) Red path: 2.5 years old female buffalo collared in point a in South Africa in October 2013 walked for a maximum direct distance of 95kms, in 6 days. She crossed into Zimbabwe then Mozambique (point b) and in Zimbabwe again to enter GNP where she entered the HR of a buffalo herd collared between 2008 and 2010 (point c). She was visually sighted (by plane) on the 23rd of January 2014 within a 70-strong mixed buffalo herd in the southern part of GNP (H. van der Westhuizen, pers. comm.). Note the straight line followed by ID65 inside GNP representing the railway line crossing the park and entering Mozambique; 2) Purple path: a 4-years old young female buffalo collared in October 2013 in point e (and initially captured but not equipped in July 2011 at 20 months) walked at a direct distance of 64kms. She joined the northern tip of the path in 8 days, where a small buffalo herd is suspected to range (point f); 3) a 4.5 years-old female, captured (point g) in June 2010 at 24 months, was re-sighted in March 2013 96kms direct

distance (points h), from its capture site, deep into communal land (B. Lessmay, pers. comm.). Note that the three events end out of the TFCA green (Caron *et al.* 2015b).

This behaviour of young female buffalos is quantitatively demonstrated here for the first time and has implication for buffalo ecology and conservation. First, we believe this behaviour to be an outbreeding behaviour before reproduction during the season when resources are not limiting factors, increasing the likelihood of success. However, we do not know if these individuals move alone or in groups. The fact that we did not observed any adult female engaging in such behaviour over our large dataset makes us believe that adult females are not involved in this behaviour. But the open question is how these females knew where to go and when (experience individual in the group or clue in the environment?). These results indicate that the Sengwe Corridor is already functional at least for buffalos (and elephants has it has been shown by other study). So do we need to remove the communities living in this corridor or not? Our data should inform decision makers. Our results raise also the question of the boundaries of TFCAs, supposed to be ecologically-based but clearly not for young female buffalo who cross them!

These new observations coupled with an in-depth review of available literature on the ecology of the African buffalo led me to redefine the concept of a buffalo herd (Figure 46) as a build-in concept influenced by a set of different factors favouring fusion or fission of core buffalo groups.

Figure 46: Proposal for a new buffalo herd concept



A buffalo "mixed herd" is composed of 1 or more "basic herds", the smallest group unit where female buffalos occur. Around mixed herd gravitate bachelor groups according to current definition. A mixed herd can range from thousand plus individuals when several basic herds mix to a few individuals belonging to only one basic herd. On top are external factors that contribute to the fission-fusion dynamics of buffalo groups. For example, as habitat heterogeneity increases, mixed herds tend to

split in basic herds. At the bottom is a summary of the risks and benefits and their effects on basic herd decision to stay or leave mixed herds (Cornelis et al. In prep).

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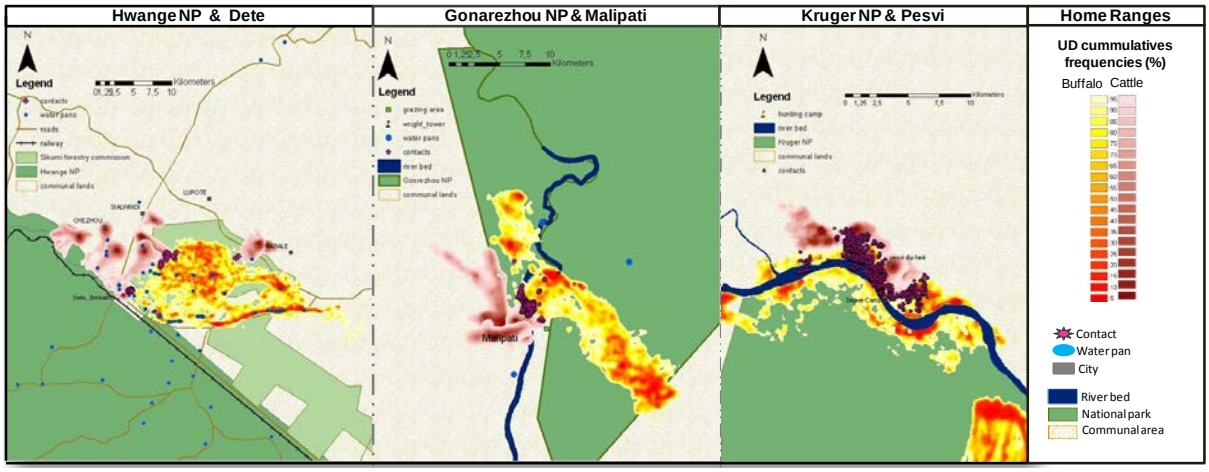
I will not present the details of this on-going work but just say that 1) it could help us testing hypotheses related to the size of groups and human disturbance; 2) This framework has important implications in terms of disease transmission within buffalo population and between buffalo and other species' populations (see below). I hope that we should be able to test these hypotheses in a near future using our large dataset on buffalo telemetry (through a PhD or a post doctorate supervision or both).

Interactions between cattle and buffalo at the population level have been studied by Eve Miguel and Fadzai Zengeya (Miguel et al. 2013b; Zengeya et al. 2015; Miguel et al. Submitted). I will only provide the broad patterns identified across the three interfaces studied (Figure 47).

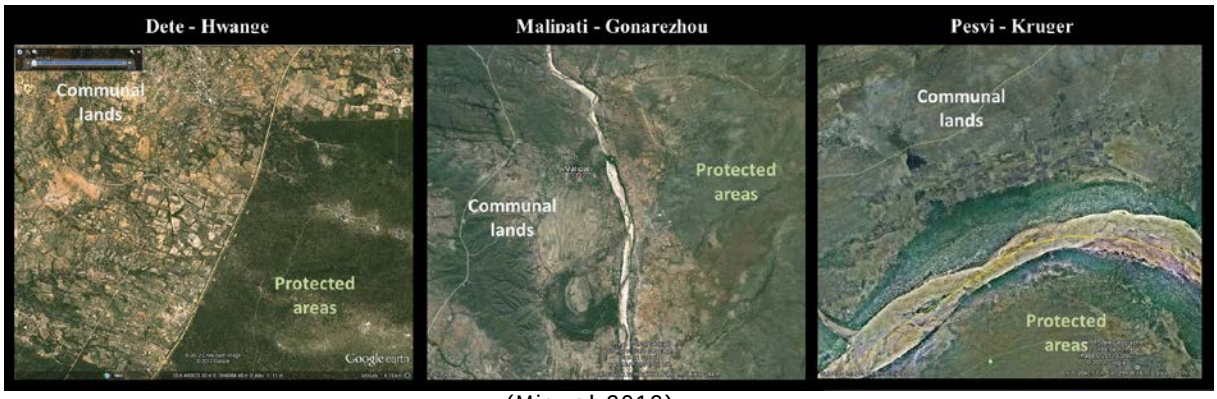
Figure 47: Home range overlap and contact (300M and 0-15 days) at buffalo/cattle interface in the three interfaces studied



(Miguel 2012)

The home range overlap between buffalo and cattle across the three interfaces is limited to a small part of the individual HR. In the HNP/Dete and GNP/Malipati interfaces, most of the overlap was inside the protected areas (in the Forestry Commission for HNP/Dete); in the KNP/Pesvi interface, most of the overlap occurred in Zimbabwe outside KNP. Displaying annual HRs does not offer the possibility to study seasonal patterns in HR overlap. In addition, HR overlap does not mean that individuals get into contacts, it only means that they use at some point the same habitat during the given time window. However we could observe that the overlap was not random in the landscape. It occurred often close to key resources such as water points and grazing areas.

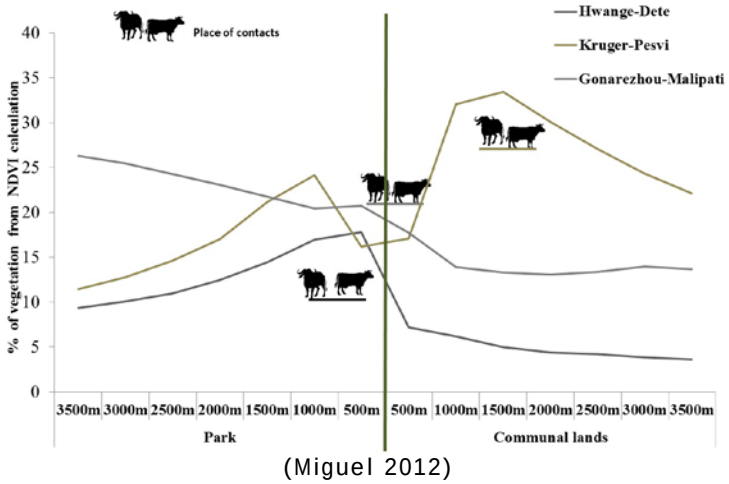
Figure 48: Google earth image of the three inetrfaces



(Miguel 2012)

To further study these interactions, one needs to define what a contact is. Telemetry data is also sensitive to some bias and we always take a buffer of 300m around the GPS location to take into account the GPS precision, the movement of the individual between 2 GPS points (30mn or 1h) and the size of the group around the focal animal (both cattle and buffalo occur in herds). A direct contact "nose to nose" has never been observed during our telemetry studies even if cattle owners mentioned seeing "buffalo grazing with their cattle". This event must be rare. Then, we can define a temporal window for indirect contacts indicating the use of the same habitat a specific intervals. In Figure 47, this time window is set at 0 to 15 days. With this definition of contacts, we clearly showed the influence of the productivity indices (NDVI) on buffalo to cattle contacts (Figure 48 & 49). The spatial distribution of key resources appear therefore to influence drastically the patterns of HR overlap. However, not taking into account cattle owners herding practices would miss some of the main factors driving cattle mobility and influencing their contacts with buffalos.

Figure 49: NDVI estimations across the three interfaces in relation to the location of buffalo/cattle contacts



(Miguel 2012)

In HNP/Dete and GNP/Malipati, cattle owners mentioned using the protected area on two occasions: firstly during the rainy season when their crops are growing and they do not want cattle to damage them; and secondly during the dry season

when grazing is scarce in the communal land and a better grazing is always available in the protected areas (Figure 49). In the two interfaces, the influence of these two practices seems to vary (e.g. the crop-avoidance behaviour was stronger in the Hwange/Dete). In the KNP/Pesvi interface, the use of the riverine forest around the Limpopo River, a good grazing area yearlong prevented the need for farmers to send their cattle in KNP during the dry season (in addition to the risk of meeting a KNP ranger squad with risks of fines or cattle being quarantined). Therefore the social component is also driving the patterns of contacts between buffalo and cattle.

Buffalo/cattle interactions & pathogen transmission

What are the consequences of the ecological interactions and behavioural patterns presented above for pathogen transmission and disease spillover?

The data presented in Figure 45 has direct implications. It indicates that buffalo populations of KNP and GNP are connected through long-distance movements of individuals, specifically pre-breeding heifers. While this is important for buffalo conservation in TFCAs, it could also facilitate the spread of animal diseases, including zoonoses, across borders. The fact that buffalo ID65 (red pathway on the figure) was seen amongst a breeding herd in GNP clearly demonstrates the possibility of direct, buffalo-to-buffalo transmission of bTB by dispersing infected individuals, without the need for bridge hosts (e.g. other wild or domestic ungulate species)(Caron et al. 2015a). We have therefore a direct answer to the hypotheses we laid down previously (de Garine-Wichatitsky et al. 2010): according to available information, we do not need the interplay of additional host to explain the spread of bTB from KNP to GNP buffalo populations as our data demonstrates that direct buffalo to buffalo contacts do occur, through specific age group and at a specific time of the year (rainy season). Of course, additional ecological information on buffalo dispersion is now required. This includes: the frequency of dispersion events, the size and age-sex composition of the dispersing groups, and whether individuals later return to their original home ranges. Sub-adult females had been infected with bTB, brucellosis and RVF (Table 15) and probably also with FMD, which are diseases with different mechanisms of transmission. Age, and social position in the herd, may also influence the rate of exposure of individuals to pathogenic infections and therefore a different risk of pathogen transmission between populations. These results indicate that sex and age class may impact on disease dynamics and, in this case, that sub-adult female buffalo could play a significant role in the spread of diseases between distant populations, across protected areas and international borders, and specifically during the rainy season. The fact that buffalo were observed far outside the boundaries of protected areas and even outside the GLTFCA, in communal land where livestock farming is the main livelihood, considerably widens wildlife/livestock interface area where disease spread can occur (Musoke et al. 2015). Thus wildlife/livestock interfaces can, in fact, encompass large areas, rather than a fence or a strip of land at the edge of protected areas. This data should therefore refine disease modelling by re-defining variables involved in the risk of pathogen spillover or emergence, with respect to age and sex of the host, e.g identifying super-spreaders)(Paull et al. 2012), as well as temporal and spatial considerations. In the case of bTB, there seems to be a gradient of spillover risk from buffalo to cattle, higher closer to protected areas (because herd to herd contacts do occur through adult females and

other individuals in mixed herds) but this risk is non-null 50kms away from the NPs through individual buffalo to cattle herd contacts. Our results suggest that the spillover of bTB and other zoonoses at the wildlife/livestock/human interface constitutes therefore a risk in the GLTFCA (Caron et al. 2013; de Garine-Wichatitksy et al. 2013a).

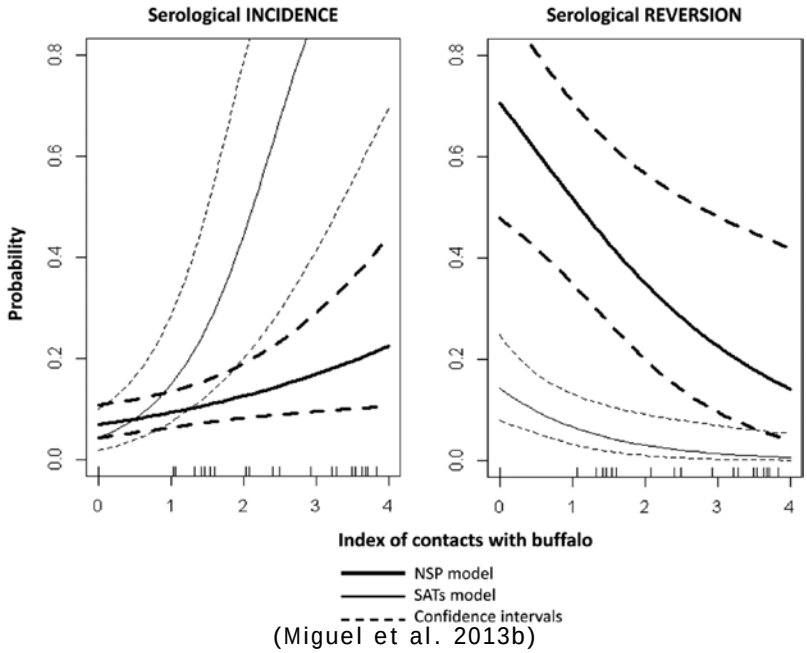
Table 15: Infection results for buffalo sampled in KNP by age groups

	Calf		Sub-adult		Adult		Total
	M	F	M	F	M	F	
Bovine tuberculosis	0/4	0/5	2/11	4/25	0/9	2/23	8/77 (4/38)
Brucellosis	0/4	1/5	1/12	4/28	2/9	5/25	13/83 (0/38)*
Rift Valley Fever	0/4	0/5	0/12	1/31	1/9	3/28	5/89 (2/38)

(Caron et al. 2015b)

In Miguel et al. (2013b), we used a longitudinal survey of FMD seroprevalence in ear-tagged cattle for more than a year to calculate the incidence of FMD in these population and to explore the relationship between this FMD incidence and cattle/buffalo contacts at the three interfaces. Contacts from buffalo to cattle were define with a spatial window of 300m (for each GPS location) and a temporal window of 0-15 days according to environmental persistence demonstrated for FMD virus under the conditions prevailing in our SES. The results prove for the first time a significant positive relationship between the number of contacts between cattle and buffalo and the incidence of FMD in cattle (Figure 50).

Figure 50: FMD serological incidence and reversion probabilities of FMD antibodies in cattle



(Miguel et al. 2013b)

These results confirm a "known fact" about the role of maintenance of buffalo and source of FMD for cattle. However, this type of quantitative relationship has always been missing to support this fact. In addition, these results are quite robust as they originate from three different interfaces. As direct contacts were almost never recorded, our results suggest that the survival of FMD viruses in the

environment is high enough for delayed contacts to generate virus spillover from buffalo to cattle. This is in contradiction with the current FMD dogma in southern Africa, in need to be further studied. Within each study site, NSP incidence (supposed to detect viral antibodies only and not vaccinal antibodies) was significantly higher at sites located close to national parks, compared to others. Lower incidence in sites far from national parks is not a demonstration of a cause to effect relationship between proximity to buffalo populations and FMD incidence in cattle because none of the other environmental variables potentially influencing FMD epidemiology was under our control. However, it supports the hypothesis that national parks harbour FMD wild host maintenance populations.

This approach exploring the relationship between buffalo-cattle interactions and pathogen transmission has the advantage to be replicable to estimate the risk of pathogen spillover one way or another (in the case presented above, we only explored the buffalo to cattle direction of transmission). The spatio-temporal window for transmission only requires being adapted to the mode of transmission of the pathogen considered. In a recently submitted manuscript, we modelled the risk of three different pathogen transmission modes at the buffalo-cattle interface, from buffalo to cattle and from cattle to buffalo (de Garine-Wichatitksy et al. Submitted). We explored contact network structures for various spatio-temporal windows defining direct or indirect contacts, compatible with interspecific transmission of important pathogens, and confront the model results with empirical serological data from the same cattle and buffalo populations.

Table 16: Characteristics of selected pathogen transmitted between buffalo and cattle

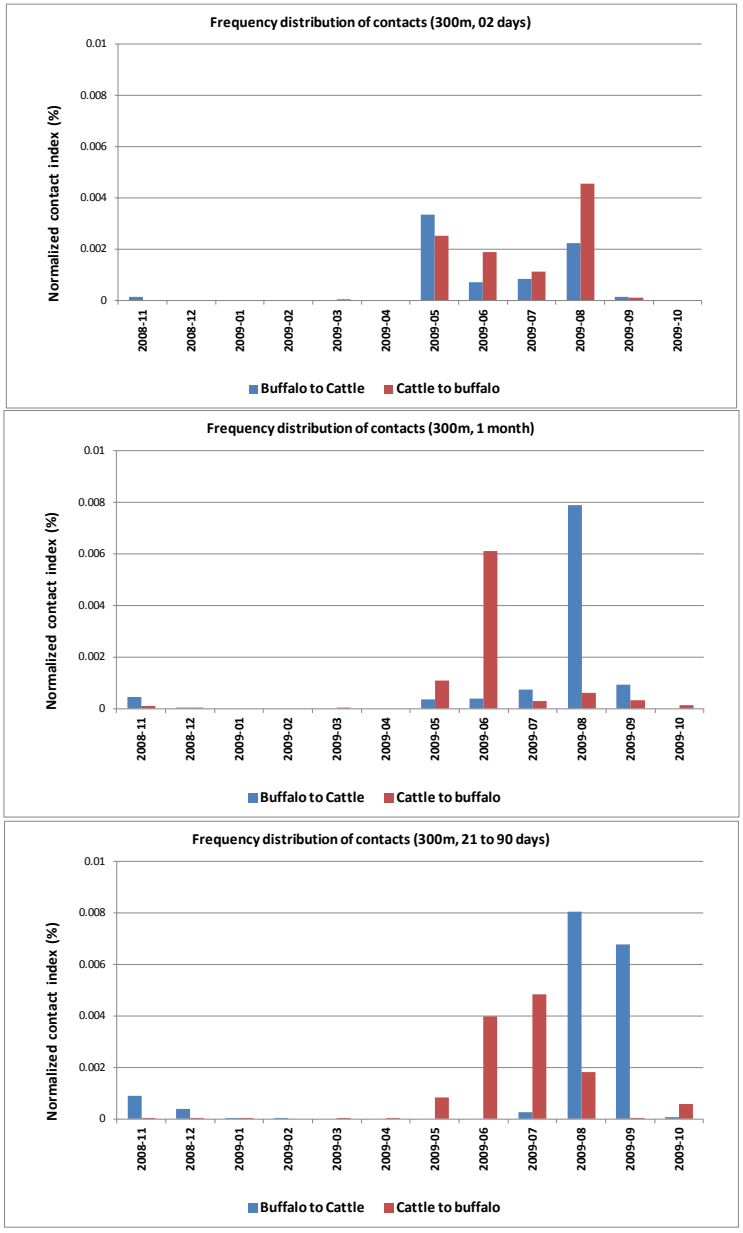
Disease name	Pathogen	Mode of transmission	Temporal window
Bovine tuberculosis	*Mycobacterium bovis*	Inhalation of aerolized droplets	0h (direct contact)
Brucellosis	*Brucella abortus*	Contact with or ingestion of infected foetus or other abortion products	0-2 days
Bovine tuberculosis	*Mycobacterium bovis*	Inhalation or ingestion of contaminated material (faeces, …)	0-1 month
Corridor disease	*Theileria parva*	Bite by infected tick vectors after feeding on an infected buffalo host	22 days-3 month

(de Garine-Wichatitksy et al. Submitted)

The distribution of contacts results indicates that (Figure 51): firstly, with increasing time windows, the number of contacts does not increase linearly and therefore the risk of pathogen inter-species spillover is not linearly related with the time window, a proxy of the transmission mode; there are less contacts for a time-window of 0-2 days than for the 2 other time windows but the difference between 30 days and 21 to 90 days is not large and the maximum probability is similar; secondly, there is an asymmetry in the risk of spread from buffalo to cattle and from cattle to buffalo (different colours in Figure 51). This of course has major implications for disease transmission. For example, we have not found any brucellosis in the buffalo population in GNP but the disease was present in all cattle populations. The risk of brucellosis spillover from cattle to buffalo is the highest during August and last only between May and August (during the year we had data at least). This means that depending on the direction of pathogen transmission of interest, the risk will vary. Thirdly, as each window models one

pathogen's mode of transmission, with the same dataset of telemetry data, the intensity and frequency of the risk of spillover for different pathogens varies and will therefore require different mitigation measures.

Figure 51: Frequency distribution of contacts between cattle and buffalo for three different time windows in relation to the mode of transmission of the three selected pathogens

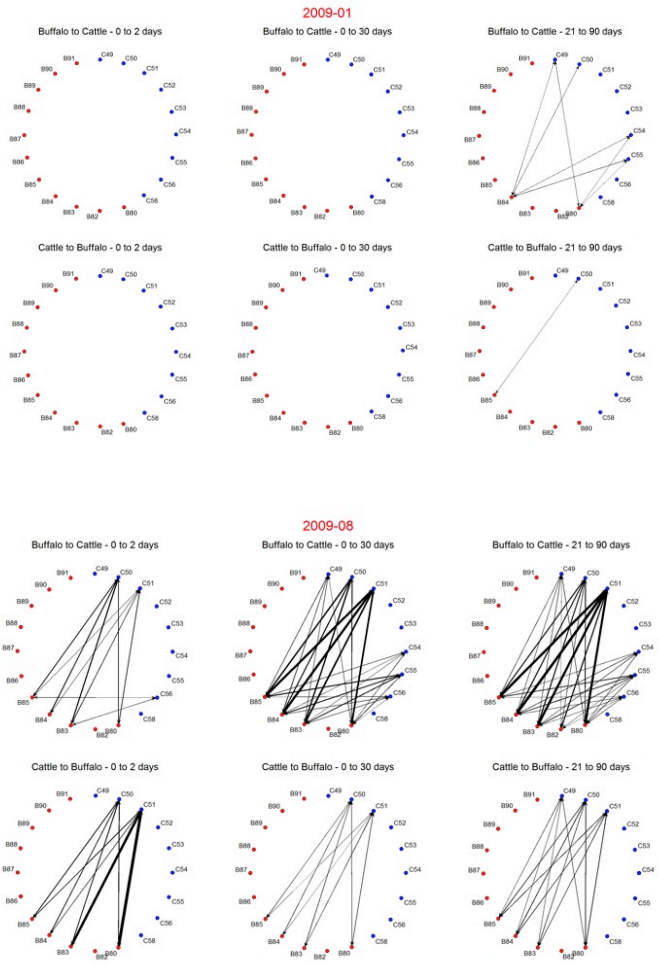


(de Garine-Wichatitksy et al. Submitted)

When we look at the same dataset through individual host network analysis where edges represent contacts as defined with the different time windows already presented (Figure 52a & b), another characteristic of buffalo/cattle contacts in our systems is highlighted: at the population level, the risk of pathogen spillover is not randomly distributed. Buffalo belonging to herd B (red nodes, lower part of the networks) have contacts with cattle nodes (blue) but buffalo belonging to herd A

have not; identically, (adult female) cattle representing the movements and contacts of the herd they belong to, do not always engage in contacts with buffalo (e.g. C52, C53) when others do (e.g. C49, C50, C51). The observations translate the different herding strategies of farmers depending on their location in the village (more or less close to the NP) but also their perceptions and decisions in relation to taking the risk of sending their cattle in the NP. These behaviours have consequences for disease transmission.

Figure 52: Network representations of individual cattle and buffalo (nodes) contacts (edges) according to the three defined time windows during January 2009 (a) and August 2009 (b)



Buffalo nodes are in red and cattle nodes are in blue. The width of the edges are proportional to the intensity of contacts (de Garine-Wichatitksy et al. Submitted)

At the individual level, buffalo belonging to herd B in contact with cattle herds, did not all engage in contacts with cattle. Buffalo B82 did not engage with cattle at all. As we have seen previously that buffalo belonging to the same herd do not always spend much time together and that fission events occur often within buffalo groups of the same herd, we can hypothesise that the use of habitat shared with cattle can be a stimuli for some buffalo to break from the core group and decide to avoid this type of landscape. Finally, the two selected month clearly show a

seasonal pattern as in Figure 51, in frequency and intensity of inter-species contacts.

Beside improving the understanding of disease ecology and pathogen transmission at wildlife/livestock interfaces in TFCAs of southern Africa (which I believe we significantly contributed to), our results can help designing targeted and innovative strategies for disease surveillance or control. Disease control measures such as fences, vaccination, and vector control, and the target of these measures in TFCAs need to be carefully considered (Bengis, Kock & Fisher 2002). First, control measures targeting livestock appear to be the least invasive for natural systems (even if acaricide control on cattle can shift tick host preferences (de Meeus et al. 2010). Second, the difficulty of applying control measures in wildlife and a lack of experience of interventions in wildlife render the outcomes of such control measures uncertain. Third, environmental control measures, such as fencing, can compromise conservation objectives such as increasing connectivity between protected areas.

FMD control in southern Africa is constrained by the poor longevity of FMD protection after vaccination (3 to 4 months estimated *in situ* by our studies). We showed that most of the contacts between cattle and buffalo leading to primary outbreaks of FMD occur at specific seasons (cold-dry and hot-dry depending of the interface) and concerned specific villages. Surveillance should focus on these areas and at the time of estimated contacts. Similarly, in order to control effectively for FMD in the area and given the fact that the country can only afford for one vaccination per year, targeted vaccination during the season at risk can help optimizing FMD control for these interface areas. For bTB, we demonstrated that the risk of spillover is not restrained to the few kilometres around the land-use boundaries but extend to a much wider area. This data can also help rethinking disease surveillance.

Finally, controlling contacts between wild and domestic ungulates can also be down through controlling the access to key resources in the landscape. Fencing is one option with its own disadvantages (see above). The management of surface water is another as we demonstrated that this key resource could explain different contact patterns between cattle and buffalo at different interfaces (KAZA TFCA interface compared to the 2 GLTFCA interfaces) and therefore influences pathogen spillover. Preventing cattle to use river pools at the interface where wildlife drink could be done by drilling boreholes at key sites within villages. These considerations imply that disease management decisions need to be debated within a framework that extends beyond a veterinary or economic perspective and should engage with all stakeholders including final users (farmers), governmental technical services and research and development experts (Miguel et al. 2013b).

F. Further work and tool development in transmission ecology

1. Application of EFG approach to other pathogen-host models

Bird pathogens & wild bird maintenance communities

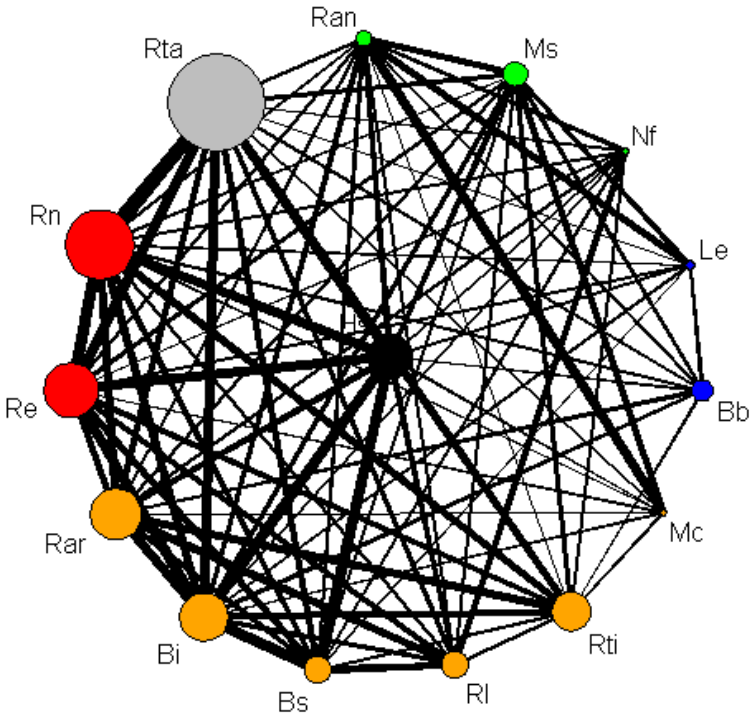
Our work on AIV in wild birds and at the interface between wild and domestic birds is replicable to other pathogen-avian models. Indeed, as our model in transmission ecology is based on identifying transmission pathways through inter-host contacts, those contacts (or their proxies) can be used to model the transmission dynamics of other pathogens, given that the modes of transmission of this pathogen are known. This approach was used for two other avian diseases, Newcastle Disease (Miguel et al. 2013a; Cappelle et al. 2015) and Avian Malaria (Cumming et al. 2013; Hellard et al. 2015).

I will only provide results for the most recent article for which I was specially asked to contribute in order to back-up the EFG approach. In this article, we used EFGs to test our understanding of Avian Malaria (Plasmodium and Haemoproteus sp.) dynamics in four communities of wetland-associated birds in southern Africa (same database as for our regional AIV studies). 176 bird species were allocated to a set of EFGs according to their assumed roles in introducing and maintaining the parasite in the system. Roles were quantified as relative risks from avian foraging, roosting and movement ecology and assumed interaction with vector species. We compared our estimated a priori risks to empirical data from 3414 captured birds from 4 sites and 3485 half-hour point counts. After accounting for relative avian abundance, our risk estimates significantly correlated with the observed prevalence of Haemoproteus but not Plasmodium. Although avian roosting height (for both malarial genera) and movement ecology (for Plasmodium) separately influenced prevalence, host behaviour alone was not sufficient to predict Plasmodium patterns in our communities. Host taxonomy and relative abundance were also important for this parasite. Although using EFGs enabled us to predict the infection patterns of only one genus of Haemosporidia, our approach holds promise for examining the influence of host community composition on the transmission of vector-borne parasites and identifying gaps in our understanding of host-parasite interactions. It has to be noted that this was the first attempt to use EFGs for a vector-borne model.

Shared community of pathogens between rodents and human

In 2009, Serge Morand gave me and Michel de Garine-Wichatitsky access to a multi-host, multi-pathogen dataset from South-East Asia (SEA) that he and his colleagues had collected recently. This was to test some of our ideas concerning EFGs that we developed further later on (Caron, Morand & de Garine-Wichatitsky 2012; de Garine-Wichatitsky, Caron & Morand Submitted). This dataset was interesting because of its richness: 17 helminth and 15 microparasite species shared between 15 sympatric rodent species and knowledge of the presence or absence in the human host. This was the perfect occasion to investigate our idea of using the community of shared pathogens between hosts to predict/infer transmission pathways within a given ecosystem or a set of ecosystems. In addition this dataset was strengthened by a habitat layer.

Figure 53: Network of shared community of pathogens between 14 rodent species and the human host (central nodes)



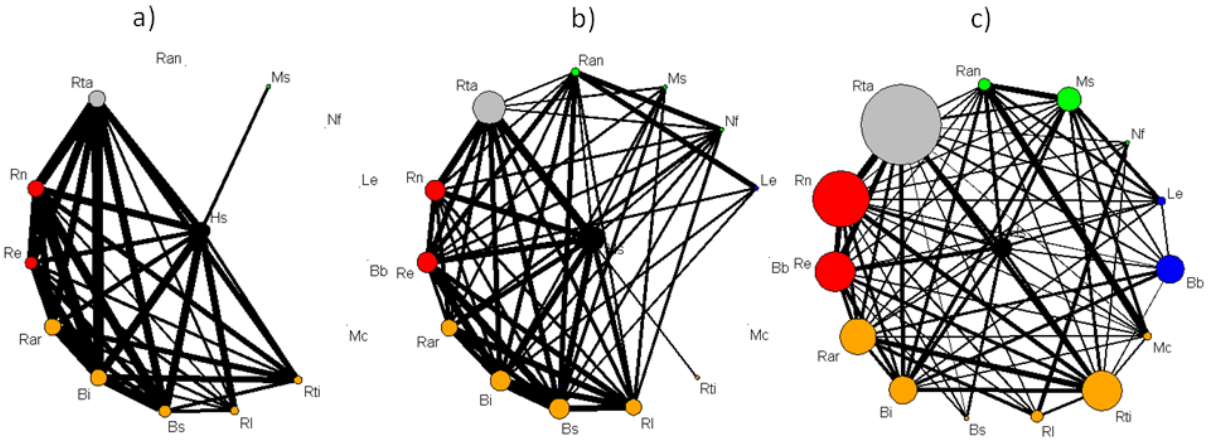
Edges are proportional to the Jaccard index matrix of shared pathogens between pairs of hosts. The human species (black node) is at the center of the network for a better visualization. Each other node represents a rodent species with its colour indicating its preferential habitat (green= primary forest, blue= highlands, orange= lowlands, red= human settlements, grey= no preferential habitat). The size of the node represents the number of parasite species detected in the host species (Caron, Morand & de Garine-Wichatitsky 2012).

The objective was to identify animal species within complex SES that are more likely to play a key role in the emergence of infectious zoonotic diseases in humans. One approach consisted in using information from current ecological and parasitological similarities between species in order to predict the most likely pathways for future pathogen spillover. We established networks based on habitat specialization, host phylogenic proximity, and infestations. Centrality was used as a proxy of the relative epidemiological importance of a species within the community of hosts. Our results emphasized that highly connected rodent species may represent a high risk of direct spill-over to humans (e.g. *Rattus tanezumi*, Rta), although they may also act as indirect bridge connecting humans with habitat specialist rodent species (e.g. *M. surifer*, Ms) potentially harbouring parasites to which humans have not been exposed yet (Figure 53). This example illustrates how network analyses may be used to improve surveillance and control of emerging zoonotic pathogens by targeting species or habitat (see on Figure 53 how rodent species specialised in specific habitat share more or less interactions with other species including the human host) that are more likely to play a significant role in future emergences (Bordes et al. Submitted).

I also tried to build the same network according to the shared modes of transmission of pathogens (Figure 54). If the sample size (number of pathogens per

category) was small, it was still interesting to observe the visual differences between the three networks. The directly transmitted pathogens were largely shared in the households or in paddy-rice fields between a defined set of rodents and the human host, while food-borne modes of transmission were involved in a denser network involving rodent hosts in all the types of habitat.

Figure 54: Shared pathogen community network between 14 rodent species and humans based on the pathogens modes of transmission



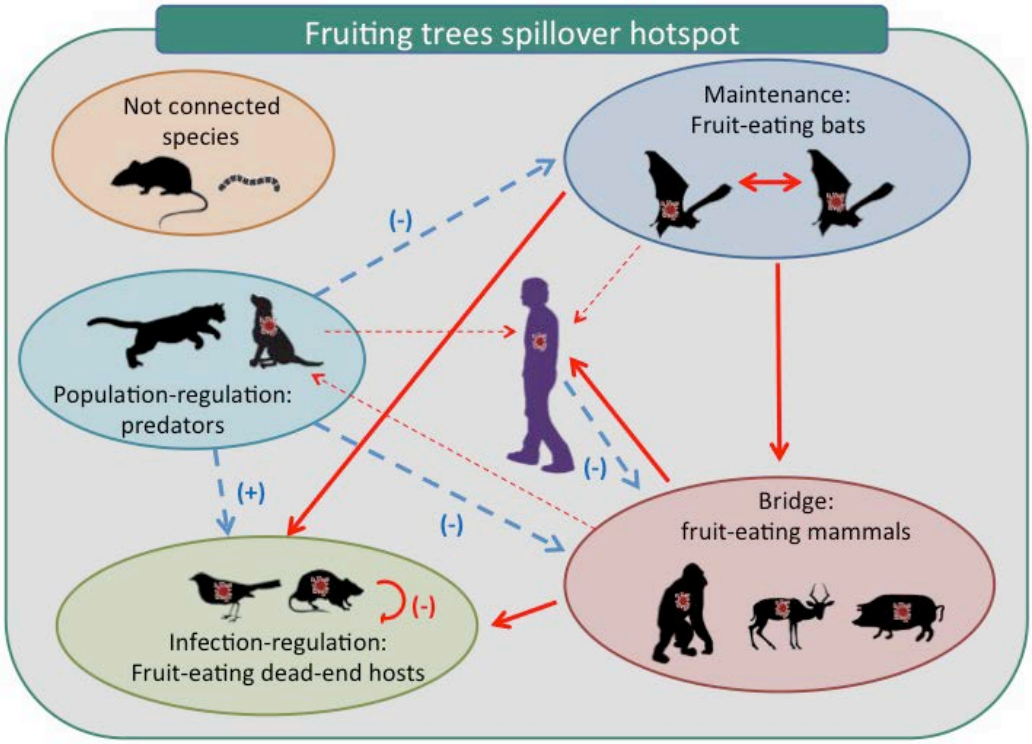
Each network regroups pathogen species with a common dominant transmission mode: a) direct transmission mode (n=6 pathogen species); b) environmental transmission mode (n=10 pathogen species); c) food-borne transmission mode (n=26 pathogen species). The target species (human, black node) is located at the centre of the network (Caron, Morand & de Garine-Wichatitsky 2012).

This type of approach can be interesting but requires large and comprehensive datasets, which are difficult to collect. In our avian pathogens or ungulate pathogens models, we do have a diversity of hosts (especially in the avian model) but we only managed to test for a handful of pathogens, preventing such analyses. In the future, I should try to set-up extensive research protocols trying to build this type of dataset in order to carry on studies on shared communities of pathogens in multi-host systems.

Ebola in a multi-host system `

Finally, to once again highlight the fact that our approaches are replicable and can be useful for other disease model, I will present an EFG model for Ebola, as this disease is relevant given the recent massive human outbreak that occurred in 2014-2015 in West Africa (Figure 55). This model was built in 2012, before the current outbreak.

Figure 55: EFG model for Ebola ecology in West & Central Africa



(de Garine-Wichatitsky, Caron & Morand Submitted)

This model is based on the current knowledge on Ebola ecology. The target host that we consider here is the human host. Note that for conservationists, it could be great apes. It is believed that the maintenance host for Ebola are bats. However, so far, too little is known about which bat species is involved in the maintenance community for Ebola. Leroy et al. (2005) have explored the transmission pathways of Ebola virus in central Africa, and suggested the following mechanisms: fruit-eating vertebrates congregate on fruiting trees, a seasonal and discrete resource in rainforests. This gathering is a potential explanation for Ebola transmission through bat saliva left on incompletely eaten fruits, dropped on the forest floor and subsequently eaten by great apes, monkeys or duikers. Human beings are thought to become infected when they eat or handle these animals. Gonzalez et al. (2007) provided serological and molecular evidence of Ebola infection of a number of wild and domestic hosts in central Africa, and mentioned a possible role played by domestic dogs in transmitting the virus from infected carcasses of wild animals to humans.

Accordingly, host species may be allocated to the following EFGs: 1) human populations in Central African forests are the target host; 2) fruit-eating bats are maintenance hosts (even if the real maintenance community is not yet known); 3) fruit-eating ground-dwelling animals are bridge host, including wild primates, some antelopes and livestock such as pigs; 4) fruit-eating dead-end animals are infection-regulation ("dilution") hosts through reduction of exposure to infected fruit material (e.g. rodents or birds not able to transmit the virus to human); 5) predators of fruit-eating host animals (e.g. wild and domestic carnivores, humans) are population-regulation hosts potentially reducing populations of maintenance and/or bridge hosts; 6) all other animals (not connected species) that do not play a role in Ebola epidemiology.

This model can help designing research or protocol surveillance. The hypothesised role of host can help choosing when and where to sample it. For example, if one wants to investigate the role of bridge host of a given host population, as this host is supposed to connect the maintenance (here bats) and the target (human) hosts, the sampling should occur in areas where the potential bridge host is in contact with human (or bats but as a departure point it seems more sensible to start by the target host). If this host is consumed, then there is an easy way to sample "cheaply" this potential bridge host by looking for it on the stands of formal and/or informal markets. Once new information is provided (e.g. the potential bridge host is a bridge host; or is a maintenance host; or does not play any role in the ecology of Ebola) then the model can be refined with new information. This seems to be a better alternative than going blindly sampling within the forest any wild ungulate or any bat species without knowing exactly which hypothesis to test. This seems obvious but this type of blind approach has been widely used in AIV ecology in wild birds when any type of wild bird was sampled for surveillance.

Therefore I believe that the EFG approach has some future to help framing research around incompletely known pathosystems and that's what I am currently doing under my involvement in the IUEPPR project aimed a better understanding the PPR ecology in Africa. I am involved in the team (with Richard Kock & Vladimir Grosbois) investigating the role of wildlife and wildlife/livestock interface in the ecology of the virus.

2. Diversity of *Escherichia coli* as a patho-indicator

At this point, I hope I made it clear that there is a need to optimise disease surveillance and control, notably in multi-host systems at wildlife/domestic/human interfaces and to adapt the surveillance tools to predict transmission and emergence before the spillover event occurs. Predicting which pathogen will emerge or spillover between hosts is challenging and a crucial step forward would be the availability of a tool aiming at identifying the transmission processes which would provide a "road map" for pathogen transmission and emergence between host populations in a given ecosystem. Such a tool would help concentrating surveillance and control towards high-risk transmission pathways where multiple pathogens could be transmitted or emerged instead of implementing multiple single host and/or pathogen surveillance, a cost-efficient way of using limited resources. Human, domestic and wild animal health could be improved by a better control of pathogen spillover between these hosts with direct benefits for rural community livelihoods, local development, biodiversity conservation and overall for the coexistence between Man and Nature.

The following presents some research I have been developing since 2 or 3 years. I have submitted proposals at the ANR Jeune Chercheur funding without success (even if the proposal went through the first round of selection several times). I developed this work first in collaboration outside AGIRs with Jean-François Cosson from CBGP (CNRS) and then with Dr. Erick Denamur and Prof. Antoine Adremont from INSERM-Hopital Bichat in Paris (contacts were made thanks to Serge Morand). I supervised two French students, Mathilde Mercat (M1) and Méril Massot (M2) and we have one manuscript resubmitted after revision (Mercat *et al.* Submitted) and another one that should be in preparation soon. I worked a lot in the last three

years to develop this research programme and hope that in a near future it will attract more funding.

How to identify transmission pathways between hosts?

The identification of transmission pathways between host populations within a SES can be investigated through different means. As I presented already, this can be done by identifying host contact patterns. It can also be achieved by using past transmission event to predict future ones. Here, the logic would go as follow: if a directly transmitted pathogen is transmitted between a pair of hosts, it means that they have direct contacts that could lead to the transmission of any directly transmitted pathogens. Then, along the same line of thoughts, the community of pathogens shared between host populations could be used, globally or using sub-groups of pathogens using similar transmission modes as presented in the previous sections (Caron, Morand & de Garine-Wichatitsky 2012; Gomez, Nunn & Verdu 2013; Bordes et al. Submitted). Finally, one can identify an indicator of pathogen transmission, a "patho-indicator" that highlights transmission pathways between hosts. This indicator would require specific characteristics to be useful (Mercat *et al.* Submitted): 1) It should be ubiquitous; 2) Well known; 3) Display a level of phenotypic or genotypic variability that could be used to track its transmission between organisms; 4) A range of tools to investigate this variability should be available and affordable; 5) Access to data should be as non-invasive as possible, especially in the case of sampling wildlife species that can be difficult and expensive.

Escherichia coli *a good candidate patho-indicator*

Based on preliminary findings (see below), we believe that the bacteria *Escherichia coli* (*E. coli*) can be such a patho-indicator. The rationale behind the use of commensal *E. coli* populations as an indicator of transmisison pathways is first based on the fact that *E. coli* is probably the best known bacteria, with numerous complete genomes of members of the species available and that its antibioresistance has also been well studied (Allen et al. 2010; Tenaillon et al. 2010). Secondly, *E. coli* populations are ubiquitous and can be found in most animal species and in the environment and are transmitted through direct, food-borne or environmental transmission (fecal-oral route) covering therefore a large diversity of transmission modes. Thirdly, *E. coli* diversity is high at the gene content level (less than 50% of the genome of *E. coli* is common to all the strains of the species) and at the nucleotide level in the conserved genes (Touchon et al. 2009). Fourthly, the population structure of commensal *E. coli* is dependant of the phylogeny of the host, its gut physiology, its diet but also of the climate and the year of sampling (Gordon & Cowling 2003; Escobar-Paramo et al. 2006). Population dynamics have been the focus of recent academic studies and technical development. Finally, investigating *E. coli* diversity and molecular properties can be done through non-invasive techniques, which is an advantage to study pathogen transmission in wildlife populations. A number of studies have recently investigated the relationship between E. coli population diversity and social proxies of contact or proxies of infectious contacts with encouraging results (Goldberg, Gillespie & Singer 2006; Goldberg et al. 2007; Goldberg et al. 2008; Johnson et al. 2008; Rwego et al. 2008a; Rwego et al. 2008b; van den Broek et al. 2009; Benavides et al. 2012; Blyton et al. 2013; Lescat et al. 2013; Pesapane, Ponder & Alexander

2013; VanderWaal et al. 2013; Blyton et al. 2014; Chiyo et al. 2014; Lupindu et al. 2014; VanderWaal et al. 2014). Antibiotic Resistance (ABR) in E. coli populations can be used as a special case of genetic diversity that can be used to track transmission. The emergence of ABR is mainly anthropogenic, resulting from a high and inappropriate use of antibiotics in humans and domestic animals (Skurnik et al. 2006). The diffusion of ABR in pristine ecosystems can be explored and used to track directional genetic transfer from human and livestock towards wildlife (Levy & Marshall 2004; Martinez et al. 2009; Allen et al. 2010). This array of facts gave us confidence that this bacteria is an ideal candidate to test our main hypothesis: "that tracking the variability of populations of non-pathogenic micro-organism such as E. coli and their genetic heterogeneity, including antibiotic resistance, allows the identification of infectious disease transmission pathways within and between host populations belonging to a host community".

Road map to explore the potential patho-indicator role of E. coli

Recently, a number of studies have investigated patterns of *E. coli* diversity in relation to proxies of contact and/or infectious transmission between single or multiple hosts. Table 17 summarizes some of the important characteristics. The results of these studies are quite inspiring. For example, Rwego et al. (2008a) used a repetitive element PCR (rep-PCR technique) to quantify *E. coli* transmission between human, primates and livestock, indicating that *E. coli* populations are shared and transmitted between interacting host populations. Lescat et al. (2013) showed that the genetic diversity of commensal *E. coli* (phylogroups) between host species can be explored at the ecosystem level. Additional recent studies include protocol comparing wild animal behaviour and *E. coli* population diversity (Pesapane, Ponder & Alexander 2013; VanderWaal et al. 2013; VanderWaal et al. 2014). If multi-host studies have already been implemented, it is clear that longitudinal studies are lacking. Only one has investigated longitudinal variability of *E. coli* population (Blyton et al. 2013), however in a single host.

Table 17: Summary of *E.coli* studies related to transmission ecology

Author	Model	Multi-host	Longitudinal data
Goldberg	Human/Primates/Cattle	X	
Johnson	Human/Cat	X	
Rwego	Human/Livestock	X	
van den Broek	Human		
Benavides	Human/Livestock/Gorilla	X	
Blyton	Brushtail Possum		X
Lescat	Human/Wild animals	X	
Pesapane	Human/Banded mangoose	X	
VanderWaal	Giraffe		
Chiyo	Elephant		
Lupindu	Human/Cattle	X	
VanderWaal	Ungulate community	X	

(Caron, unpublished data)

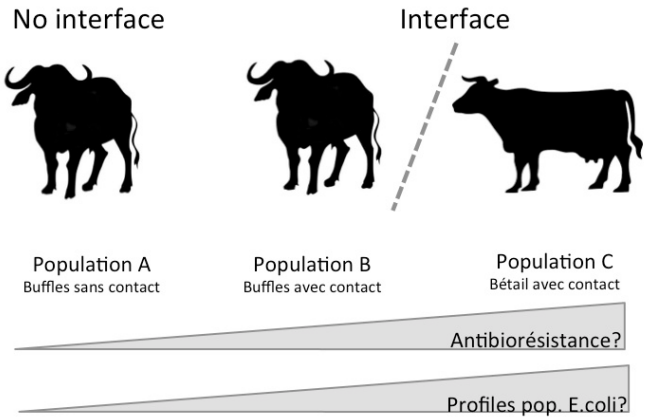
To identify and validate the use of *E. coli* population diversity as indicators of transmission pathways, one will need to follow a stepwise process that we have already started to explore through preliminary collaborations. It has to be emphasized that different indicators will be looked at for different types of *E. coli*

population variability: fine scale molecular epidemiology using whole genome sequencing, and the presence and abundance of antibioresistance genes are candidate indicators that will be tested.

The main steps are as follow:

- ***Step 1***: Prove that *E. coli* is present in the host populations studied (buffalo/Cattle/Human);
- ***Step 2***: Confirm that *E. coli* populations in the host populations studied (buffalo/cattle/Human) harbour antibioresistance genes;
- ***Step 3***: Study the variability of *E. coli* populations (including antibioresistance) at different spatio-temporal scales (e.g. spatial scale: individual, herd, village, region; temporal scale: day, week, season, year);
- ***Step 4***: Choose and adapt the molecular tools for detecting different levels of the genetic variability of *E.coli* populations and quantify it;
- ***Step 5:*** Characterize proxies of pathogen transmission to be tested against *E. coli* indicators of transmission between host populations;
- ***Step 6:*** Test if the *E. coli* indicators are correlated with the other proxies of pathogen transmission charcterised;
- ***Step 7:*** If steps 1 to 6 are sucessful, then this process should be replicated in different socio-ecosystems (e.g. urban or marine environments).

Figure 56: Conceptual model of the first *E.coli* study in the Hwange/Dete interface

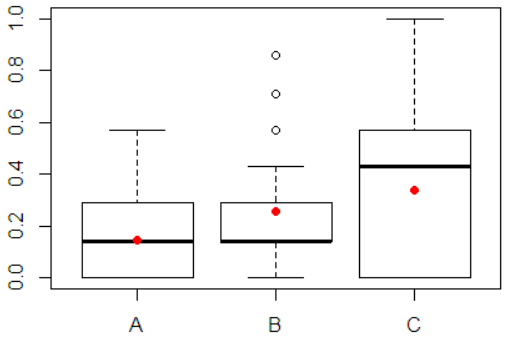


A = buffalo population not in contact ; B = buffalo population in contact with cattle population ; C = cattle population) (Caron, unpublished data)

The two MSc that I have supervised started answering Steps 1, 2 & 3. The results of these exploratory studies comforted the team's intuitions in the fact that the questions addressed were relevant, at least in the socio-ecosystem studied, and that *E. coli* populations were indeed variable, which could pave the way for more in-depth research protocols. The first study (by Mathilde Mercat) explored whether *E. coli* strains were present in sympatric cattle and buffalo populations and explored the *E. coli* phylogroup composition (Step 1) and antibioresistance (Step 2) in three ungulate populations with variable level of interaction (contact rate determined by telemetry studies)(Figure 56).

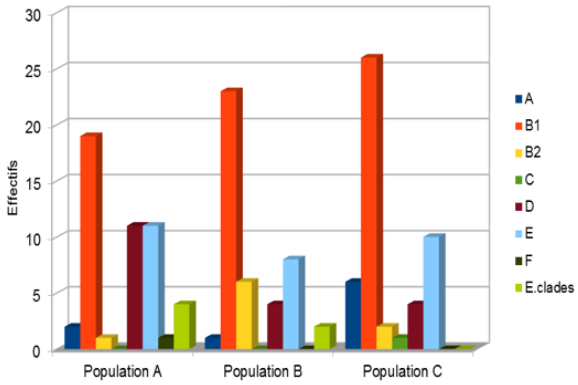
The main results of this first study were : 1) a gradient of antibioresistance observed along A<B<C indicating a potential diffusion of antibiotic resistance within the natural ecosystem (Figure 57); 2) a similarity in *E. coli* populations' composition with a majority of B1 and E phylogroup strains in cattle and buffalo)(Figure 58), which was expected because the two species are related phylogenetically and feed on the same natural ressources (water & grazing)(Mercat *et al.* Submitted).

Figure 57: Antibiotic resistance gradient between the three ungulate populations



(Mercat et al. Submitted)

Figure 58: *E. coli* phylogroup composition in each of the three ungulate populations



(Mercat et al. Submitted)

The second study (by Méril Massot now doing her PhD with the INSERM research unit of Dr. Erick Denamur) investigated the temporal variability of *E. coli* populations and antibioresistance within the cattle host (part of Step 3). Four heads of related cattle from the same herds in Magoli village were sampled daily during 4 weeks at fixed hours with faecal swabs taken at the same time from the four individuals. Analyses carried out at the INSERM laboratory in Paris on the samples measured at: 1) the global pattern of antibiotic resistance as in Lester et al. (1990); 2) the genetic diversity and antibiotic resistance of dominant clones of *E. coli*; and 3) the proportion of the main *E. coli* phylogroups as in Smati et al. (2013).

The main results indicate a certain stability both in terms of diversity and abundance of *E.coli* populations within individuals (Figure 59). However some change in dominant phylogroups and abundance seems to be related to environmental change such as dipping (cattle are treated with acaricides once or twice a month to prevent tick-borne diseases).

Figure 59: Abundance (top) and diversity (bottom) of *E. coli* strains for each head of cattle sampled every day during one month

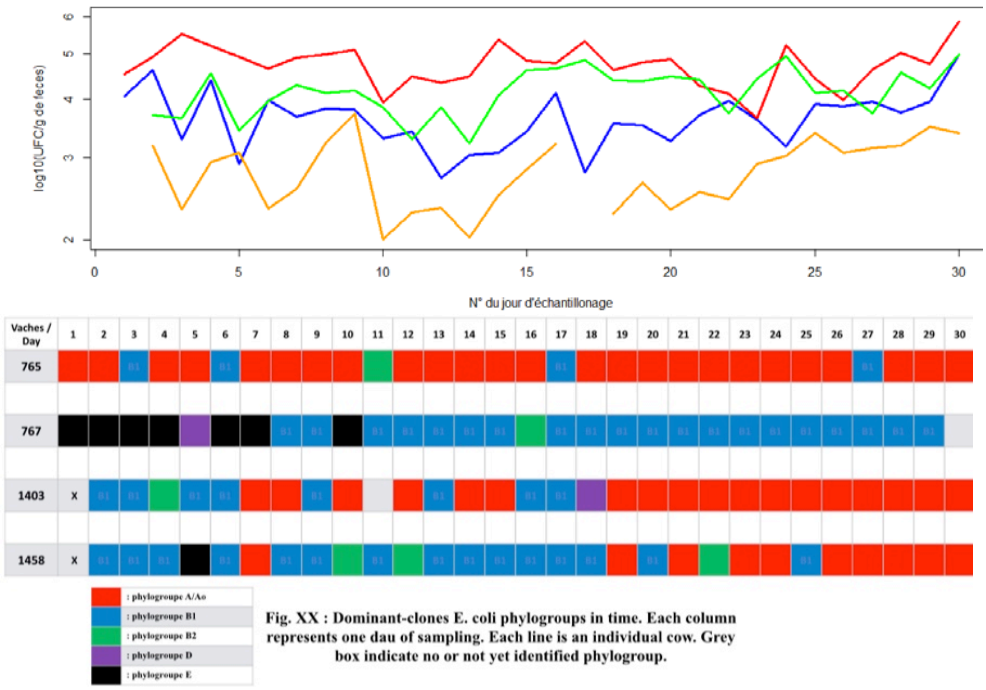


Fig. XX : Dominant-clones E. coli phylogroups in time. Each column represents one dau of sampling. Each line is an individual cow. Grey box indicate no or not yet identified phylogroup.

In the first graph, each colour represent a different head of cattle; in the second graph, each line represetns a head of cattle and colours represent the dominant E.coli strains isoalted for each day (Massot et al. unpublished data)

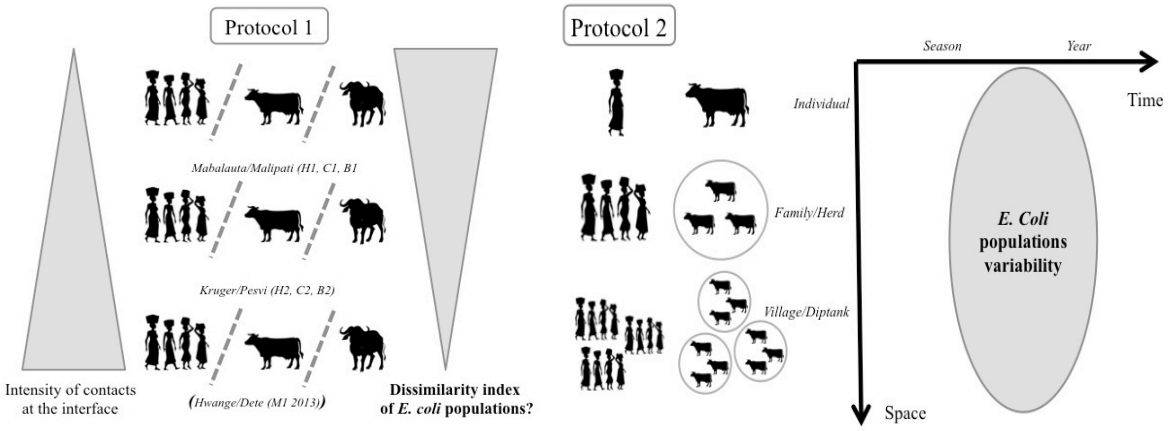
These studies have highlighted a similarity in *E. coli* phylogroup structures between buffalo and cattle sharing space and resources (based on telemetry studies implemented on both species). They also identified a decreasing gradient in antibiotic resistance spreading from cattle to buffalo populations in contacts and to buffalo populations not in contact with previous populations, suggesting a transfer of antibiotic resistance from human to cattle and then to buffalo (based on the type of antibiotic use in each population)(Mercat *et al.* Submitted). Finally, the latest study on cattle, pinpointed the variability of *E. coli* genetic structure in cattle at a short time-scale indicating at the individual level, variability in *E. coli* counts at the daily level but some consistencies in the phylogroups and antibiotic resistance identified. Both studies have helped us understanding at which molecular scale to focus our attention and using which molecular tool to detect the variability in *E. coli* population structure, which will constitute the primary matter for our future investigations.

This is where I am now. The objective is to proceed along the road map. If Step 4 is a purely molecular step in the research agenda that needs to be tackled by my collaborators, Step 5 will rely on the on-going works in the selected study sites and new data collected. The different proxies of pathogen transmission which will be used are: (a) Host contacts: telemetry studies on buffalo and cattle (Miguel et al. 2013b; Caron et al. 2015b); (b) Observed shared pathogen(s) between hosts (Caron et al. 2013); (c) Exposure to infectious transmission risk by human practices (including husbandry) and quantification of its frequency and intensity; this data can be collected using participatory tools; d) finally host population genetic can infer the level of genetic flow between species populations; currently, a population

genetic study is investigating this in buffalo populations in the area (already mentioned in an earlier section (Smitz et al. 2013; Smitz et al. 2014).

We have designed three protocols to be implemented in our TFCAs' study sites that could help progress along the roadmap and answer Steps 1, 2 & 3 (Figure 60). The first one will compare the variability of *E. coli* diversity across the three human/livestock/wildlife interfaces that we have been studied. The second one would explore *E. coli* diversity in space and time in livestock and people.

Figure 60: Protocols to further explore the role of *E. coli* as a potential patho-indicator



(Caron, unpublished data)

If funded we should be in a position to test if *E. coli* diversity and population genetics can be used as a neutral indicator of transmission pathways in a given multi-host system and if more research should be invested in this research area. If yes, we will have developed a set of bacteriological and molecular tools to characterise the diversity of *E. coli* populations and its antibioresistance within individual samples. Implications could be important for veterinary and public health surveillance if we manage to assess where/when pathogens (including emerging unknown ones) have the highest probability to spillover between host populations. Results could also be important in terms of biodiversity conservation as the spillover of antibiotic resistance into the wild has unknown consequences.

Some of these last pieces of research may seem too much drifting towards fundamental research in place of more applied research as is usually implemented within the framework of research for development promoted by Cirad. I believe that this research has its place along side with more applied research. I have presented our work within African TFCAs where we implement applied research on animal diseases and the transmission of pathogens at wild/domestic interfaces. This research feeds the knowledge about local dynamics and the role of different hosts in disease ecology and informs decision makers (local such as farmers or national such as technical services). This applied research also gives the opportunity to develop in parallel but also in complementarity (see how ecological and epidemiological works assist in testing the *E. coli* as patho-indicator study) more fundamental works. *In fine*, this fundamental work (e.g. *E. coli* study or rodent-SNA) is not completely disconnected from reality as, if successful, could lead to improve disease surveillance and control.

G. Brainstorming on my future research

1. Identification of my future research challenges

As I am just starting a new expatriation in Mozambique based at the Veterinary Faculty of the Universidade Edouardo Mondlane, Maputo, it seems timely to reflect on the research I would like to develop and to identify what I need to achieve this research. In addition, since 2015, I have taken more responsibilities in the coordination of the RP-PCP and this will require that I locate my research within the broader SES approach.

My future research will be built on the framework that I have presented here: applying the EFG approach to multi-host systems at wild/domestic interfaces. First, the theory that we have proposed needs to be further developed and conceptualised. Second, it also needs to be tested in other multi-host patho-systems. I am currently involved in a PPR (peste des petit ruminants) project where we are exploring the role of wild ungulates in disease ecology at wild/domestic interfaces in East Africa. We have already with Vladimir Grosbois, Nicolas Gaidet, François Roger and Richard Kock (RVC) revisited a sero-database dating from the PACE project (Pan African Control of Epizootics) and tried to assess the performance of serological tests for PPR in wildlife species in order to assess the true prevalence in these species and inform and orientate today's surveillance in wildlife, trying to identify the role of wildlife species in a maintenance or other type of EFG. My research unit is also starting projects or writing-up proposals on Ebola ecology in animals and at the animal/human interface or on informal bush meat trade in Africa involving the related sanitary issues. I hope that I will be able to get involved in the design of research protocols and implement EFG framework-driven research.

Using commensal bacterial transmission and antibiotic resistance to identify transmission pathways between populations is a theoretical field of research that I would like to keep on developing as it supports well the EFG approach. However recent failures at attempts to obtain funding on this topic despite having an excellent consortium supporting my initiative have decreased my motivation. This being said, antibiotic resistance diffusion at human/livestock/wildlife interfaces is currently a hot topic and maybe with this angle I could access funding more easily with, as a second objective, the patho-indicator research question.

Being involved in comparative studies brings additional emphasis and insights on research results. Our approaches across 3 wildlife/livestock interfaces to study disease burden in TFCAs or across multiple study sites in Africa for AIV in wild birds triggered important comparative research and results. I will need therefore to be associated with large multi-site projects to reach again this level of outputs and analysis. This could be done within AGIRs through bridging our two "Dispositif de Recherche en Partnariat" RP-PCP & GREASE or through the collaboration with other institutions. I have good contacts with some colleagues from the University of Liverpool whom I met at several occasions and with who I share research interests. They already operate in Kenya and we discussed the potential to write project proposal together. Given the amount of ecological data (telemetry and biodiversity data) that we have collected so far (in Zimbabwe, South Africa, Mozambique and potentially other countries through the IUCN AfBIG group on the African buffalo

that I coordinate with Daniel Cornelis), we can also start implementing meta-analysis through PhD and post-doc opportunities. With Daniel Cornelis and Michel de Garine-Wichatitsky, we have written a Phd proposal on buffalo behaviour that we will try to submit for funding (55% of the funding is now secured at least). If accepted, I should be one of the main supervisors. Identically we could submit a post-doctoral proposal trying to link telemetry data and transmission ecology, modelling animal behaviour and relating it to the risks of pathogen transmission at different types of wildlife/livestock interfaces.

I will need also to develop more research on the proxies of pathogen transmission between hosts. The two broad domains concerning this issue are characterising gene/genetic material flow between host populations and host contact patterns. The recent development of Next Generation Sequencing (NGS) and the amount of power of genetic and molecular analyses that is now available create an opportunity to bridge more transmission ecology and molecular tools. Host or pathogen genetics can shed light on pathogen transmission dynamics or contact between host populations (both with a spatial and temporal dimension). This area is not new but every year new technologies supersede previous ones, decoupling the capacity to produce genetic data from individual samples. This is what I tried to develop in the "*E. coli* as a patho-indicator" project by bringing in a research team specialised in *E. coli* genetics and population dynamics. Those NGS tools can also more easily build database on the diversity of pathogens within host individuals, populations and communities and should allow testing hypotheses impossible to test until now.

Behavioural ecology (such as the work we have implemented on cattle and buffalo) is important to understand host contacts. But to properly describe what drives potential infectious contacts at wild/domestic interfaces, one needs a deep understanding of what drives domestic animal movements and behaviour. This is only partly related to behavioural ecology and requires understanding on what influences herding strategies. Participatory tools and social sciences' (anthropological) approaches are therefore necessary to access farmers' values, perceptions and their resulting decisions in terms of farming practices. These variables are difficult to access without skilled professionals and their techniques.

From a more applied perspective (and in line with Cirad applied research mandate), I would be interested also to work on the design of optimised surveillance systems at wildlife/livestock interfaces. Such functional surveillance systems would improve livestock production in communities neighbouring protected areas, would better protect wild species from emerging disease and could also be the first line of defence against zoonoses in human populations. The objectives of this type of surveillance system should be to: 1) be negotiated with all stakeholders (farmers, veterinary and national park services etc.) and should respond to all stakeholders expectation, facilitating the buy-in of all; 2) ensure an open flow of information about morbidity and mortality events in wild and/or domestic populations; 3) be cost-efficient by providing cheap information transmitted by motivated stakeholders. For example, in Mozambique, veterinary services have very weak extension services in arid and semi-arid areas and information as well as veterinary knowledge would need to percolate both ways from the field to central chain of command and vice versa. There is a need to think about local animal surveillance systems using opportunities such as new rapid diagnostic tools or the

extensive coverage of cellular networks that can promote communication of sanitary and emergency information much faster. Coupling such surveillance systems with livestock monitoring systems (tracking numbers, productivity indices such as fertility etc.) would also provide relevant information for all stakeholders. One of the areas that I have started to explore (in relation to my PPR activities) is the potential to develop disease sampling from hunted wild animals. There are thousands of animals shot for trophy hunting or for rations by park or concession staff. With the advance in technologies to detect antibodies but also pathogen genetic material from specific types of filter paper, the capacity to obtain good quality samples from the field (no need for a good cold chain) is increased and such a system extended across a country could provide important epidemiological data to better understand infectious disease shared at wildlife/livestock interfaces.

Within the framework of GAH (Global Approach to Health), I believe I should also extend more my research to the Animal/Human interface. In the SES where I work, public and veterinary services both suffer from a lack of means to complete their mandate. In addition to the economy of scale and sharing of information (on zoonoses mainly), there is a need to understand more the impact of zoonoses on human health and how it can be surveyed (on animals? on humans?) and controlled. Diseases such as brucellosis, rift valley fever, bovine tuberculosis are usually not detected in human because they are classified as "malaria-fever syndroms" as no option for diagnosis are available. The optimization of surveillance systems could therefore also be extended at the Animal/Human interface.

Finally, as mentioned during the first chapter of this manuscript, the relationship between Biodiversity and Health (of animals and humans) an the questions around the resilience of SES notably in relation to Health are still largely underexplored and I hope that I could contribute to the discussion through my research and my ideas.

However, this "liste à la Prévert" can only be useful if I have the skills and expertise to work on these topics. As this research is related to very different fields of research (ecology, epidemiology, social sciences and others), I can only pretend to master some of them. In the next sections, I will present my skills, those that I intend to acquire and those that I will not master and will therefore need to seek through collaborations.

2. Skills that I would need to develop and those that I will not

Knowing one strengths and weaknesses is the beginning of success. I pretend to know my weaknesses. In fact, as I see myself more as a generalist (the future rare asset of science I believe) than a specialist, I have many. For example, I can hardly say to be a specialist in any disease/infection at wildlife/livestock interface. Yet I have contributed to publications on avian malaria & influenza, bTB, FMD, Newcastle disease, PPR, brucellosis, tick-borne diseases including theileriosis and others, Ebola viruses, lumpy skin disease, antibiotic resistance, rinderpest, rodent-borne diseases and RVF.

Therefore, there are many skills that I would need to master in order to achieve the research agenda that I have presented above: biostatistics (e.g. Bayesian); modelling (e.g. basic SEIR models but also more complex embedded models with some spatial components or population dynamics modules); social sciences

(including participatory sciences and approaches), genetics and tools to analyse molecular analyses; population dynamics (e.g. bacterial, mammals); behavioural ecology and in particular telemetry data analyses.

Of course I will not be able to master them all. But, I see a generalist as a multi-plug researcher with a main "male" plug connecting to its principal body of research "Ecology of disease transmission in multi-host systems"; "female" plug are multiple and should encompass all of the skills presented above. What I mean by female plug is having enough understanding of this field to be able to connect to it and use it to feed my own research questions, to know what can be done with its data and tools. I have followed trainings on most of these topics (biostatistics, population dynamics, social sciences) and have a basic understanding of how they can help me testing my research questions.

More practically, I need more and more training on biostatistics, my main weakness, maintained mainly because I don't use them on a regular basis. If I keep on working on *E. coli* as a patho-indicator I would need more advanced training on population dynamics applied to microorganisms. More training on telemetry analyses would be also important even if so far my colleagues have done this for me. In addition, I need additional exposure on most of the other topics just to understand better how I can use them. Specifically, I would need to work on some collaboration with modellers on projects/research questions (and I have many) to understand well how far can we explore epidemiological processes and test EFG hypotheses using models. Finally, for most of these skills/topics, the use of the software "R" is now recommended. I have a basic knowledge of how to use "R" but I would need more training on programming in "R".

Definitely, I will never master epidemiological modelling, genetics, and social sciences and will need to seek additional collaboration on these fields.

3. My Dream Team

Therefore, as I am a generalist and only capable of mastering a few aspects of the expertise I need to answer the research questions I am interested in, I need to be surrounded by a team of colleagues (within Cirad/Research Unit) and collaborators (outside Cirad) who fill my gaps in knowledge and skills. Of course this framework centred on myself is purely an artificial construction of this HDR manuscript; in reality, there is no centre but a sum of researchers centred on their research questions (male plug) and connected/contributing to other researchers through their female plugs.

The following gathers a list of skills that I would need to have within the first circle of my research team:

- The R geek: in order to explain to me the range of possible in terms of biostatistics; to help me in doing statistics and programming in R; to develop epidemiological models. This skill is largely present within AGIRs.
- The Geneticist: it seems that one needs more than one person for this skill as there are specialists of hosts (mostly mammals) and specialists of pathogens. This field is in constant evolution and growing almost exponentially. The level of expertise to analyse the amount of data is also impressive. This skill is present within AGIRs but outsourcing these skills

(for example as I have done for my *E. coli* work with the INSERM-Bichat team) is still a necessity.

- The Anthropologist: as I am more and more discovering the field of social sciences in general and its power to unravel the crucial articulation between people perceptions (a consequence of their history, socio-cultural experience etc.), their behaviour and their impact on processes of disease transmission, mirroring the contribution of ecology to understand wildlife behaviour and its impact on infectious contact with domestic and human hosts. This skill is present within AGIRs and Cirad.
- The Microbiologist: because my work starts at the host population level but I rely on individual diagnostic tests to understand infection at the population level. A specialist of host-pathogen interaction within the host and of the reliability of diagnostic tests is therefore necessary. This is not present within AGIRs but within CMAEE, a Cirad research unit with which our unit could merge in the next years.
- The Ecologist: an ecologist of the host (e.g. mammal ecologist) can shed more light on behaviours and the design of protocol adapted to these behaviours. Or even adapt domestic animal or human behaviour through the prism of ecology, which sometimes can be particularly relevant. This skill is present within AGIRs
- The Project/Development Manager: as I am a Cirad researcher, someone needs to keep its feet on the ground and make sure I am not flying to high into theoretical clouds. I partly have this skill but always noticed that I benefited to have someone more focused on these things beside me. This is often a luxury but in a Dream Team, this position is important.
- Another Disease Ecologist: because it is important not to be always embedded within the same research framework, to discuss, exchange and share and produce new ideas.

These skills/positions can be filled by the same person or by students sometimes. Of course, this list represents the first circle of collaborations needed to implement my research. As a Cirad researcher hosted in a foreign institution abroad, the local matrix of stakeholders, technical services (e.g. veterinary services), NGOs, local authorities and the final beneficiaries of our research, the small-scale farmers are all crucial to integrate research within the local needs.

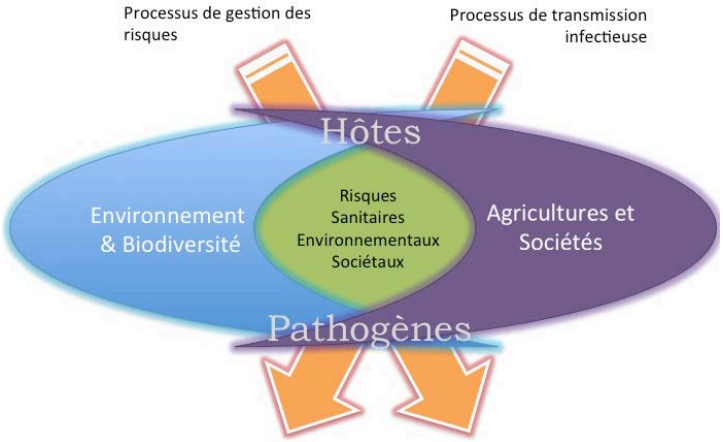
4. Future research agenda

To conclude this non-altruist exercise that made me concentrate for hours on MY research and before going back to more collaborative research work, I tried to synthetize here, at the end of the journey, what would be my research agenda in the coming years.

Firstly, this agenda needs to fit within my research unit agenda. In 2015, I have been heavily involved in the request by Cirad direction to merge our research unit with the CMAEE UMR. Without giving too much details for those unaware about Cirad history, I am just providing a scheme that I have produce for the new research unit and which represents quite well the fine balance that needs to be found between (more fundamental) research and development (through research), the two pillars promoted by Cirad. My research is today more focused on the

"infectious transmission processes" of infectious pathogens and diseases. I would like during this next phase to develop more my contribution to the other aspect around the "management processes" including as mentioned previously some work on surveillance and monitoring systems. This could be done in collaboration with my colleagues within the AGIRs UPR.

Figure 61: Proposed research framework for the new research unit on Health, Animal, Human, Biodiversity, and Environment within Cirad



(Caron, unpublished data)

Secondly, geographically, I see southern Africa as my home base for research with extensive experience in its region. However, I intend to extend my region of activity. As Mozambique has also a step in Eastern Africa, the second region in Africa where I have the most experience, I foresee some potential collaboration with research networks in Tanzania for example, and maybe in Kenya and Zambia (e.g. PPR currently spreading from Tanzania to Zambia and Mozambique). I also have a foot now in the Indian Ocean region where many pathogens and contexts are shared with the continent and which could offer interesting semi-experimental protocols in comparative studies. The fact that France is present in this region (Reunion Island, "Iles Eparses", Mayotte) and that new collaboration are emerging between the Indian Ocean and southern African regions (both within the SADC community) can help in the development of research between the regions.

Finally, as I am now coordinating a Dispositif Prioritaire du Cirad and in charge of linking this DP with another one in the Indian Ocean (DP "One Health-Ocean Indien"), I will logically develop my research in strong relationship with the RP-PCP research agenda, "promoting the co-existence between Man & Nature in TFCAs". This agenda does not address the main problems of countries or populations but target some of the most vulnerable human populations trapped in their development trajectory by conservation and poor national support and services. The health (in its broad sense) of these populations will impact positively or negatively the resilience of the socio-ecosystems where they live, which are key in the success of the conservation and development agenda in Africa.

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